National Library of Canada
Acquisitions and Bibliographic Services Branch
395 Wellington Street
Ottawa, Ontario
K1A 0N4

Bibliothèque nationale du Canada
Direction des acquisitions et des services bibliographiques
395, rue Wellington
Ottawa (Ontario)
K1A 0N4

Your file Votre référence

Our file Notre référence

NOTICE

The quality of this microform is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us an inferior photocopy.

Reproduction in full or in part of this microform is governed by the Canadian Copyright Act, R.S.C. 1970, c. C-30, and subsequent amendments.

AVIS

La qualité de cette microforme dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de qualité inférieure.

La reproduction, même partielle, de cette microforme est soumise à la Loi canadienne sur le droit d'auteur, SRC 1970, c. C-30, et ses amendements subséquents.

Canada

UNIVERSITY OF ALBERTA

STUDIES IN PAIN PERCEPTION, DISABILITY AND PHYSICAL THERAPY IN LOW BACK PAIN

©

BY

MAUREEN JANET SIMMONDS

A THESIS

SUBMITTED TO THE FACULTY OF

GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE

DOCTOR OF PHILOSOPHY IN REHABILITATION SCIENCE,

FACULTY OF REHABILITATION MEDICINE

EDMONTON, ALBERTA

SPRING 1994

National Library of Canada
Acquisitions and Bibliographic Services Branch
395 Wellington Street
Ottawa, Ontario
K1A 0N4

Bibliothèque nationale du Canada
Direction des acquisitions et des services bibliographiques
395, rue Wellington
Ottawa (Ontario)
K1A 0N4

Your file Votre référence

Our file Notre référence

The author has granted an irrevocable non-exclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of his/her thesis by any means and in any form or format, making this thesis available to interested persons.

L'auteur a accordé une licence irrévocable et non exclusive permettant à la Bibliothèque nationale du Canada de reproduire, prêter, distribuer ou vendre des copies de sa thèse de quelque manière et sous quelque forme que ce soit pour mettre des exemplaires de cette thèse à la disposition des personnes intéressées.

The author retains ownership of the copyright in his/her thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without his/her permission.

L'auteur conserve la propriété du droit d'auteur qui protège sa thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.

ISBN 0-612-11369-8

Canada

UNIVERSITY OF ALBERTA

RELEASE FORM

NAME OF AUTHOR: Maureen Janet Simmonds

TITLE OF THESIS: STUDIES IN PAIN PERCEPTION, DISABILITY AND PHYSICAL THERAPY IN LOW BACK PAIN

DEGREE: DOCTOR OF PHILOSOPHY

YEAR THIS DEGREE GRANTED: SPRING 1994

Permission is hereby granted to THE UNIVERSITY OF ALBERTA LIBRARY to reproduce single copies of this thesis and to lend or sell such copies for private, scholarly or scientific research purposes only.

The author reserves other publication rights, and neither the thesis nor extensive extracts from it may be printed or otherwise reproduced without the author's written permission.

(signed)
Date: Dec 30 19 93

Permanent address:
School of Physical Therapy
Texas Womans University,
1130 M.D. Anderson Blvd.
Houston, Texas. U.S.A.
77030-2897

UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled, " STUDIES IN PAIN PERCEPTION, DISABILITY AND PHYSICAL THERAPY IN LOW BACK PAIN", submitted by Maureen Janet Simmonds in partial fulfilment of the requirements for the degree of Doctor of Philosophy.

Dr S Kumar
(Supervisor)

Dr E Lechelt
(Co-supervisor)

Dr S Hunka
(Co-supervisor)

Dr T Guidotti

Dr J Vargo

Dr M Pope

Date: December 3, 1993

To: Faculty of Graduate Studies and Research
University of Alberta
Edmonton

In regards to the doctoral thesis by Maureen Simmonds entitled: Pain perception, disability and physical therapy in low back pain, we hereby give permission for our names to appear as co-authors of articles, whether published or not.

Signed: S Kumar

Dr S Kumar

E Lechelt

Dr E Lechelt

WESTFÄLISCHE
WILHELMS-UNIVERSITÄT
MÜNSTER

Institut für Experimentelle Biomechanik
GESCHÄFTSF. DIREKTOR: UNIV.-PROF. DR. RER. NAT. P. BRINCKMANN

Universität Münster · Institut für Experimentelle Biomechanik
48129 Münster

48129 Münster, Dec 1, 1993
Hausadresse:
Domagkstraße 11
48149 Münster
Telefon: Vermittlung (02 51) 83-1
Telefax (02 51) 83-59 83
Telex 8 92 529 UNIMS d
Telefon: Durchwahl (02 51) 83-59 88

Dr. Maureen Simmonds
School of Phys. Therapy
Texas Woman's Univ.

Dear Dr. Simmonds,

I am not aware that you need my permission (if so: it is herewith granted), but rather the permission of the publisher: Springer Verlag, Vienna.

Sincerely

Paul Brinckmann

Your Ref

Our Ref DJM/SJS

Date 20 December 1993

Suffolk College
Hope Walk Ipswich Suffolk
United Kingdom IP4 1LT

Telephone (0473) 255885
Fax (0473) 230054

Principal
Derek Mortimer OBE BSc MEd

Vice Principal
David Hall MSc PhD ARCS DIC FInstP

Vice Principal
Professor Dave Muller BEd PhD C.Psychol FBPsS FRSA

FAX: 010 1 713 794 2162

Dr Maureen Simmonds
Texas Woman's University
Houston Center
UNITED STATES OF AMERICA

Dear Maureen

Many congratulations. There is no need to seek permission to include your published paper as part of your PhD dissertation. If however, it is to be published commercially you will need permission from Taylor and Francis who are responsible for publishing *Disability and Rehabilitation*. I can arrange this for you if required.

Yours sincerely

Professor Dave Müller
VICE PRINCIPAL (ACADEMIC)

TOTAL P.01

ELSEVIER
SCIENCE PUBLISHERS B.V.

Dr. Maureen Simmonds
School of Physical Therapy
Texas Woman's University
1130 M.D. Anderson Boulevard
Houston, TX 77030-2897
U.S.A.

Direct Line: (020) 5862 833

Amsterdam, 11 October 1993

Dear Dr. Simmonds,

Thank your for letter of October 4th 1993, copy herewith enclosed, requesting permission to reproduce the following articles, published in the **International Journal of Industrial Ergonomics**, in your doctoral dissertation:

"Health Care Ergonomics. Part I: The fundamental skill of palpation - A review and critique" Vol. 11, pp. 135-143 (1993) and

"Health Care Ergonomics. Part II: Location of body structures by palpation - A reliability study" Vol. 11, pp. 145-151 (1993)

We are pleased to herewith grant permission to reproduce this material, provided that you give full acknowledgement to the original source of publication, and that your work is not distributed commercially.

Yours sincerely,
ELSEVIER SCIENCE PUBLISHERS B.V.
Academic Publishing Division

Jan van den Heuvel
Rights & Permissions

Encl.

TEXAS WOMAN'S
UNIVERSITY
DENTON/DALLAS/HOUSTON

SCHOOL OF
PHYSICAL THERAPY
1130 M.D. Anderson Blvd.
Houston, Texas 77030-2897
713/794-2070

Elsevier Science Publishers B.V.
P.O. Box 103,
1000 A C Amsterdam,
The Netherlands,

4th October, 1993

Dear Sir/Madam,

Simmonds MJ, and Kumar S. Health Care Ergonomics. Part I: The fundamental skill of palpation - A review and critique. **International Journal of Industrial Ergonomics** 1993, **11**: 135-143

Simmonds MJ, and Kumar S. Health Care Ergonomics. Part II: Location of body structures by palpation - A reliability study. **International Journal of Industrial Ergonomics** 1993, **11**: 145-151

With reference to the articles noted above I would like to request permission to include a version of these papers as a chapter in my PhD Dissertation. I will of course acknowledge the fact that the copyright is held by Elsevier Science Publishers and that permission for reproduction was obtained, should this be granted. For your information this work was conducted during my PhD studies at the University of Alberta. My PhD is in Rehabilitation Science.

Thanking you in anticipation. I look forward to a reply at your earliest convenience.

Yours sincerely,

Maureen Simmonds MSc PhD Cand.
Assistant Professor

TEXAS WOMAN'S UNIVERSITY

DENTON/DALLAS/HOUSTON

SCHOOL OF
PHYSICAL THERAPY
1130 M.D. Anderson Blvd.,
Houston, Texas 77030-2897
713/794-2070

Jules Rothstein PhD PT
Editor, Physical Therap
APTA,
1111 North Fairfax Stre
Alexandria,
Virginia,
22314

4th October, 1993

APTA, 1111 North Fairfax Steet,
Alexandria, VA 22314-1488

Permission to reprint is granted with the understanding that 1] no charge for profit is made other than to redeem the cost of reproduction [reprints for book publication excluded] and 2] all duplicated material carry the notation: "Reprinted from_____ with the permission of the American Physical Therapy Association."

K Quantrille
Karin Quantrille
Director of Publications

10/15/93
Date

Dear Sir,

Simmonds MJ, Kumar S, and Lechelt E. Quantification of forces and resultant motion during spinal motion testing and treatment by physical therapists using a spinal model. **Physical Therapy** 1993, Submitted

The article noted above is presently in the review process and copyright privileges have been transferred to the Journal. I would like to request permission to include a version of this paper as a chapter in my PhD Dissertation. I will of course acknowledge the fact that the copyright is held by APTA and that permission for reproduction was obtained, should this be granted. For your information this work was conducted during my PhD studies at the University of Alberta. My PhD is in Rehabilitation Science.

Thanking you in anticipation. I look forward to a reply at your earliest convenience.

Yours sincerely,

Maureen Simmonds MSc PhD Cand.
Assistant Professor

DEDICATION

This work is dedicated with love to the three most important men in my life. To David, my husband and my best friend. To Chad and Kent, my young sons who make me feel so loved, and of whom I'm so proud.

ABSTRACT

The conservative management of low back pain (LBP) frequently involves physical therapy. Many different treatment approaches are used, but most have not been empirically tested. All treatments are based on clinical assessment findings, thus it is necessary to determine what factors influence assessment findings. The purpose of this thesis was to evaluate some specific but fundamental assessment skills utilised by physical therapists (PTs). And to test whether experimental pain tests may aid in the evaluation of patients with LBP.

The first experiment was aimed to determine whether prior knowledge of compensation status influenced judgements of physical impairment or disability prognosis. In a randomised controlled study, 69 PTs viewed videotaped assessments of LBP patients. Prior to viewing the videotape the PTs were provided with a medical history of the patient which included a notation that the patient was (WCB), or was not, receiving compensation (NWCB). A third group was provided with no information on the patient (CON). The type of information did not influence physical assessment findings such as range of motion. But, patients who were thought to be WCB claimants were judged to have a worse prognosis of disability and less likely to benefit from therapy compared to those who were not WCB claimants. This implies that the expectation of outcome may influence the actual outcome. The purpose of the second experiment was to assess the accuracy of assessment findings based on static palpation. The influence of the form and depth of a structure was investigated using an invisible skin marker. Twenty PTs participated in this test-retest experiment. The location of superficial structures was found to be less accurate if the structure was irregular in shape, such as the posterior superior iliac spine (PSIS); or was deeper, such as a vertebral transverse process. There was significant variability in individual skill of PTs. A third experiment investigated motion palpation using a manufactured model. Ten PTs participated in this experiment. Manually applied forces and the resultant motion were quantified in this study. Individual variability of skill was again found. In addition, PTs systematically underestimated the amount of force that they used during manual assessment and treatment and overestimated the motion that occurred. The final experiment was aimed to determine whether clinical assessment of LBP could be enhanced with the use of experimentally induced pain. Twenty patients with LBP were compared with 20 pain free controls. Pain threshold was measured using mechanical stressors and with controlled spinal loading. No differences were found between groups on mechanical induced pain measures. However spinal loading to pain threshold was lower in the LBP group and was sensitive to change in clinical status, as was walking speed. These latter two tests are recommended as outcome measure of treatment efficacy.

ACKNOWLEDGEMENTS

It was a humbling experience to write acknowledgements for this thesis. The work herein, was a result of the assistance and the cooperation of many people. I gratefully and humbly thank them all.

I would particularly like to acknowledge the significant role played by the following people.

My first thanks go to Dr Roger Scudds, co-advisor and mentor during my Masters degree. Roger was a constant source of knowledge and encouragement. This encouragement as well as his belief in me and in my abilities, led to my pursuit of doctoral studies.

My major thanks go to Shrawan Kumar, advisor, mentor and true friend. His wisdom and sensitivity and his ability to think big were inspirational. There will always be a special place in my heart for Shrawan. A man with a great mind and a huge heart. Thank you Shrawan, I will miss you.

To Dr Gene Lechelt my co-advisor, whose initial support was instrumental in the approval of this special PhD. His valuable advice, encouragement and moral support leaves me indebted. I would particularly like to acknowledge the helpful advice prior to interviewing for academic appointments. Thank you, your advice paid off handsomely.

To Steve Hunka, co-advisor and awe-inspiring statistician. Thank you for your expert advice, for your time and especially for your patience with the statistically challenged. I feel priveliged to have received your help.

To Dr Jim Vargo and Dr Tee Guidotti committee members, thank you for your support, for your time and for your perceptive and invaluable feedback.

I feel priveliged to be able to extend my thanks to Dr Malcolm Pope external examiner from the University of Vermont. Thank you for taking the time to involve yourself with this work. I appreciate your comments, advice and support. Your signature gives prestige to this first PhD thesis in Rehabilitation Science.

Finally, to the clinicians, students, and patients, with whom I have worked over the years and from whom I have learned so much - thank you.

TABLE OF CONTENTS

CHAPTER PAGE

1. INTRODUCTION

Overview of the thesis 1
The neuroanatomical and neurophysiological bases of LBP 1
Psychosociomedical bases of LBP and disability 1
Physical Therapy and LBP 1
Influence of knowledge in the assessment of LBP 2
The fundamental skill of palpation 2
Pain and the placebo in rehabilitation 2
Spinal motion testing and treatment 3
The relationship between experimental pain, clinical pain and function 3
General discussion 3
References 4

2. NEUROANATOMICAL AND PATHOPHYSIOLOGICAL BASES OF LBP

The extent of the problem 5
The anatomy and physiology of LBP 6
Structures producing pain 7
The intervertebral disc 7
The facet joints 10
Neural basis of pain 12
Nerve root 12
Dorsal root ganglia 13
Dorsal horn 14
Conclusions 15
References 16

3. PSYCHO-SOCIO-MEDICAL BASES OF LOW BACK PAIN

Introduction 22
Psychological predictors of LBP 24
The "pain-prone masked depressive" 24
Depression 24
Cause or consequence 25
Families and pain 27
Locus of control and coping strategies 28
Socioeconomic and iatrogenic factors in disability 31
Compensation, pain and disability 32
Summary 34
References 36

4. INFLUENCE OF KNOWLEDGE IN THE ASSESSMENT OF LBP

Introduction 46
Methodology 48
Overview 48
Physical therapist observers 48
Videotaped subjects 48
Information packages 48
Videotape procedure 50
Experimental procedure 50
Observation procedure 50
Data analysis 51
Results 51
The effect of INFO 51
Correlations between variables 52
Discussion 57
Physical assessment 57
Impairment and disability 58
Prognosis of disability 58
Summary 60
References 61

5. THE FUNDAMENTAL SKILL OF PALPATION: A REVIEW AND CRITIQUE

Introduction 64
Palpation in health care 64
Static palpation 65
Summary 69
Motion palpation 69
Summary 72
References 74

6. LOCATION OF BODY STRUCTURES BY PALPATION: AN EXPERIMENTAL STUDY

Introduction 78
Method 79
Sample 79
Experimental design 79
Tasks 79
Testing postures 79
Procedure 79
Equipment 80
Analysis 80
Results 82

Discussion 89
Conclusion 90
References 92

7. **PAIN AND THE PLACEBO IN REHABILITATION**

Introduction 94
The placebo and coping 96
The extent of the placebo effect 97
TENS, laser and placebo 99
TENS 100
Laser 101
Discussion 101
References 104

8. **SPINAL MOTION TESTING AND TREATMENT**

Introduction 110
Method 112
Experimental design 112
Subjects 112
Tasks 112
Equipment 113
Procedure 114
Analysis 114
Results 117
Discussion 123
Force 123
Displacement 124
Perception of force and motion 125
Conclusions 126
References 127

9. **THE RELATIONSHIP BETWEEN EXPERIMENTAL PAIN, CLINICAL PAIN AND FUNCTION**

Introduction 130
Purpose 131
Method 132
Sample 132
Equipment 135
Procedure 138
Data analysis 139
Results 139
Group differences 139
Change over time in the experimental group 140

The effects of gender 140
Quality of pain 140
Disability and experimental pain 142
Relationship between experimental pain measures 148
Discussion 151
Clinical pain and disability 153
Quality of pain 153
Gender 154
Conclusion 154
References 155

10. GENERAL DISCUSSION

Overview 160
Physical therapy assessment of patients with LBP 160
References 165

LIST OF TABLES

TABLE		PAGE
4.1	Characteristics of the videotaped subjects	49
4.2	Group means (percentage of normal) and standard deviations of ROM for each subject	54
4.3	Group means and standard deviations of gait posture and SLR for each subject	55
4.4	Group means and standard deviations of impairment, disability and prognosis for each subject	56
6.1	Descriptive statistics of mean distances between intra-rater consecutive palpation tests	86
6.2	Descriptive statistics of mean distances between inter-rater consecutive palpation tests	87
6.3	Results of post hoc t-tests between mean intra- and inter-rater distances for each location	88
8.1	Descriptive statistics of peak applied force for each grade of mobilization and each level of stiffness	118
8.2	Descriptive statistics of peak displacement for each grade of mobilization and each level of stiffness	119
8.3	Means of therapists assessment of spinal model for simulation of physiological motion and vertebral stiffness	120
9.1	Subject characteristics	133
9.2	Clinical pain characteristics of the experimental group at initial assessment	134
9.3	Means and standard deviations of experimental pain threshold measures for males and females	141
9.4	Means and standard deviations for pain and disability measures in patients with sharp vs. dull pain	143
9.5	Pearsons correlation coefficients between pain threshold measures in the control and experimental group	149

9.6 Pearsons correlation coefficients between clinical pain and disability measures in the experimental group 150

LIST OF FIGURES

FIGURE		PAGE
6.1	Marking pen and ultraviolet light	81
6.2	Illustrates the illumination of marks with the light	83
6.3	Illustrates the illumination of crosses made in inter-rater test	84
8.1	Schematic of spinal mobilization model	115
8.2	Photograph of therapist applying a mobilisation technique to the spinal model	116
8.3	The perceived and actual magnitude of applied force for each grade of mobilization in the minimum stiffness condition	121
8.4	The perceived and actual magnitude of motion for each grade of mobilization averaged across stiffness conditions	122
9.1	Pressure dolorimeter	136
9.2	Forceps algometer	137
9.3	Mean pain threshold measured with the dolorimeter for each group, on each location and on each occasion	144
9.4	Mean pain threshold measured with the forceps algometer for each group, on each location and on each occasion	145
9.5	Mean pain threshold measured with controlled spinal loading for each group, on each occasion	146
9.6	Mean walking speed for each group, on each occasion	147

CHAPTER ONE

INTRODUCTION

Overview of the thesis

This thesis is comprised of a series of papers which both review and study aspects of low back pain (LBP), and its management by physical therapy. Each paper or chapter is complete within itself, but each contributes to the total work. The aim of this introductory section is to summarise each of the following chapters and illustrate the links between them all.

The neuroanatomical and neurophysiological bases of LBP (Chapter 2)

LBP is generally perceived as a health problem of epidemic proportions. But what is the extent of the problem? Chapter 2 briefly outlines the epidemiology of LBP, before embarking on the main purpose of the paper. That is, to review the anatomical and neurophysiological aspects of low back pain specifically as it relates to pain from the disc and facet joints, and to neurophysiological aspects that augment nociception.

Although anatomical and neurophysiological factors can explain neurological activity which provides nociceptive input, Nociception is not pain. The perception of pain and the pain experience is much more complex than nociception. The pain experience is greatly influenced by emotional, cognitive and social factors which are discussed in the next chapter.

Psychosociomedical bases of low back pain and disability (Chapter 3)

Psychosociomedical factors influence the effect of low back pain. They help to determine whether LBP is perceived as a threat or a challenge and whether it leads to total disability or causes little disruption in life. The purpose of Chapter 3 is to review those psychosocial factors that have the potential to influence the extent of disability due to LBP. In the psychological domain, these factors include anxiety, depression, coping skills and belief systems; in the social domain, factors such as family support and role models may influence disability. In addition, medical factors such as the extent and the appropriateness of medical treatments; economic factors such as compensation, all have the potential to influence the outcome of LBP and are thus reviewed.

Physical Therapy and LBP

As noted earlier, a multitude of factors in many different domains influence pain perception and the behavioural effects of pain. They also influence whether professional health care is sought, and even whether it is appropriate and effective. Despite the fact that 80% of episodes of acute LBP

will resolve spontaneously, many patients consult a physician and are then frequently referred for physical therapy (Deyo, 1983). Physical therapists (PTs) thus play a major role in the conservative management of LBP. Given the present financial crisis in health and social welfare systems and the increasing rate and thus costs of disability due to LBP, it is encumbant upon all health care professionals to ensure that the health care that they provide is both necessary and helpful.

There is a lack of empirical support for many techniques and treatments used in the conservative management of LBP. Therefore part of this thesis is aimed to evaluate some of the fundamental assessment and treatment techniques utilised by PTs. Chapters 4 - 8 are critical reviews and experimental studies which aim to do this.

Influence of knowledge in the assessment of LBP (Chapter 4)

Prior to any treatment a patient must undergo an assessment. Specific assessment findings determine the treatment plan and are used as a basis for prognostic judgements. At the present time, the objectivity of assessments and prognoses are not known. The purpose of the study reported in Chapter 4 was to determine the objectivity of assessments and prognoses in LBP patients.

The fundamental skill of palpation (Chapters 5 and 6)

The study of physical therapy judgements alluded to above, is limited in that the research protocol did not allow any physical touching of the patient. Physical therapy is fundamentally a tactile profession and valuable information is said to be gained through palpation. The skill of palpation was critically reviewed and tested in order to determine the usefulness of this skill. Palpation is usually divided into static and motion palpation. Neither technique has been subjected to adequate scientific scrutiny. The study presented in Chapter 6 is a test of the accuracy of static palpation and a determination of physical factors which influence the accuracy of location of key body structures.

Pain and the placebo in rehabilitation (Chapter 7)

Once an assessment is completed PTs have many treatment skills at their disposal aimed to alleviate pain and dysfunction. These include: electrical, thermal, mechanical and light modalities; manual therapy and therapeutic exercise. Many techniques and treatments in common use have not been adequately tested for efficacy. The fact that symptoms resolve has been accepted as proof of the specificity of treatment effects. This belief ignores the fact that most symptoms resolve spontaneously with no treatment. It also fails to acknowledge the fact that all treatments have both specific and non-specific (or placebo) effects. Many myths surround the placebo; amongst them is the myth that the placebo effect is of fixed magnitude (Wall, 1992). In reality the effect varies greatly and is dependent on many different factors. These factors pertain to the patient, the clinician, the specific outcome measured as well as

the treatment. The theoretical basis and the factors influencing the placebo effect are reviewed in Chapter 7. In addition, the magnitude of the placebo response is explored in relation to the use of TENS and laser for pain relief.

Spinal motion testing and treatment (Chapter 8)

A treatment method in widespread use for spinal problems is manual therapy. Similar to other PT treatment techniques manual therapy does not have a strong scientific base. The specific mechanisms of effect have not been determined, partly because little is known about the characteristics of the manually applied forces used in this treatment. A spinal model was designed and constructed, in order to quantify the characteristics of the manually applied forces and to determine the degree of motion that would occur under different conditions of stiffness. In addition, the perceptual accuracy of PTs was measured. This study is presented in Chapter 8.

The relationship between experimental pain, clinical pain and function (Chapter 9)

It is important to evaluate the judgements and treatment skills of therapists. Therapists are required to make judgements about the patients pain and disability. However, it is essential to determine how pain and disability are judged by the patient with the problem and whether there is agreement between the PT and the patient. The study presented in Chapter 9 addresses this issue. In addition, this study addresses the problem of the persistence of pain, whether there is a change in pain perception and if so, how this change in pain perception effects disability.

General Discussion (Chapter 10)

It was explained above that the purpose of this introductory chapter was to weave the links between the individual studies and provide the rationale behind the thesis. However it is also necessary to discuss the results of the studies within the framework of this thesis and in relation to the literature. This discussion and the conclusions based on this body of work is presented in Chapter 10.

REFERENCES

1. Deyo RA. Conservative therapy for low back pain. Distinguishing useful from useless therapy. Journal of the American Medical Association 1983:250;(8);1057-1062

2. Wall PD. The placebo effect: an unpopular topic. Pain 1992:51; 1-3.

CHAPTER TWO

NEUROANATOMICAL AND NEUROPHYSIOLOGICAL BASES OF LBP[1]

The extent of the problem

Low back pain (LBP) is a significant problem in all industrialised countries around the world (53). It is responsible for over 2 million adults consulting their general practitioners each year in Britain (3). It is estimated that the lifetime prevalence of LBP is 80% (2). Gender does not seem to affect the frequency (1). Although prevalence rates of LBP are similar between industrialised countries, the management of LBP differs. For instance, frequency of surgery varies between countries and even between genders. The United States has the highest rate of surgery. One hundred and fifteen thousand laminectomies and 34,000 other surgical procedures are carried out each year (1). Surgery is twice as frequent in men as in women and the average age for surgery is 42 (1). Despite the different emphasis in the management of LBP the long term results of surgical or conservative management are comparable (57). Neither have any significant long term beneficial effect. And after all, 90% of acute LBP will settle spontaneously (52).

Regardless of the method of LBP management, the financial burden of LBP is enormous. The fact that LBP strikes during the working years contributes to the costs associated with LBP. In 1988 Statistics Canada reported that of all compensation injuries in Canada, LBP constituted 27% (63). Kumar et al (33) estimated that in Alberta alone the WCB spent more than $113 million for the year 1983. The costs of LBP have also been increasing. It is interesting and disturbing to note, that the rate of increase is much greater for LBP than for other conditions (1). Also, the rate of disability due to back pain has increased at a rate 14 times greater than the population growth (20). However, in work related injuries, 10% of the claims are responsible for 80% of the costs of back injuries (62).

Apart from the financial costs of LBP, there are also human costs. LBP is said to be the most common cause of activity limitation in persons less than 45 years of age (31). Occupational surveys from the United Kingdom indicate that 25% of all working men are affected by low back disorders each year, and one out of 25 workers change their job due to back problems (52). Tollison (67) reported that 80% of patients attending a Pain Clinic had complaints of back pain. This implies that the pain of LBP is a major problem. Dixon (17) has suggested that back pain is as much a problem of pain as it is a problem of the back.

---

[1]A version of this chapter has been published. Simmonds & Kumar 1992. Neuro-Orthopaedics 13: 1-14

What is the nature of the pain of LBP? Chronic back pain has been rated at the same level as cancer pain (40). In addition, causalgic pain which may be a feature of LBP is one of the most severe forms of pain (40). What is the relationship between tissue injury and LBP? And why is LBP so disabling? The multidimensional nature of pain coupled with the complex and intricate structure of the spine, serve to make the answers to these questions enigmatic. Despite the many advances made in the area of LBP, fundamental questions such as that posed by Kellgren (30) and Mooney (43) "where is the pain coming from" remain unanswered. What are the mechanisms which cause LBP, and are they the same mechanisms as those which prolong it? It would seem likely that the mechanisms which cause LBP are primarily anatomical and physiological. There is really no evidence to suggest that LBP stems entirely from the "mind". However, the pain experienced and the dysfunction that results is primarily a function of the individual's psyche and motivation, and of their social milieu. Research data support the importance of psychosocial factors in the prognosis of disability due to back pain (5,38). It is also apparent that monotony, stress, and low job satisfaction contribute more to the incidence of LBP than the physical loads that the spine is subjected to (21). It has been established that in general emotional disturbances are a consequence rather than a precipitator of chronic pain (49). In addition, the duration of pain leads to a greater influence of psychological factors (25). This should not be surprising as chronic pain is a very severe stressor (6).

Thus the problem of LBP is multidimensional and research aimed at addressing the problem rightly emanates from many disciplines within the basic and clinical sciences. A comprehensive literature review of all dimensions of LBP requires a series of papers. The focus of this paper is to review the literature in terms of the focus on nociceptive aspects of LBP, the pain of LBP also requires psychological input in terms of perception, effects, disability, coping etc. The anatomical and physiological literature has been reviewed with a view to addressing the following questions: Where is the pain coming from? Why does it hurt so much? Why does it persist? And why does it lead to so much disability?

The Anatomy and Physiology of LBP

Although LBP is more than just a problem of the back, the structure and function of the back is a large part of the problem. It is useful therefore to review some relevant anatomical aspects of this structure, in order to determine the potential role of the structure in LBP. Bogduk (7) has posited the axiom that in order for a structure to be a potential source of pain it must be innervated. The implicit assumption in this axiom, is that the innervation is nociceptive with receptors responsive to mechanical or putative chemicals at their terminals or along their axons. However, given that the axiom is correct, how helpful is the premise? It turns out that all spinal structures are richly innervated this includes the bones, the facet joints and their associated

capsules, ligaments, muscles, nerves and blood vessels. In addition, many structures have a multiple nerve supply which is multisegmental. For example, the facet joints are supplied by two (8,24) or three (74) successive spinal nerves. Only the inner aspects of the intervertebral disc and the posterior aspect of the dural sheath appear to lack innervation (7,74). However, this fact does not preclude the initiation of nociception from these structures due to the presence of nociceptive fibres and putative chemicals in the vicinity. Thus discrimination of specific painful tissues is fraught with difficulties due to the rich and often multisegmental innervation of virtually all tissues.

In summary, it can be stated that pain can potentially be generated from many spinal tissues, either directly or indirectly. It is also evident that definitive statements in regards to the specific offending structure can not be made due to the "tightness" of the spinal structures and the abundant and intimately related innervation patterns in this area. In addition, pain patterns from various structures overlap (30,74). The degree of overlap from specific structures will vary individually, and the degree of overlap will also vary according to the intensity or severity of the pain.

All spinal structures have been implicated in contributing to LBP, albeit there is no universally accepted diagnostic and classification scheme for LBP at the present time (19). A variety of diagnostic labels are appended to the syndrome of LBP. These are based on specific structures eg. discogenic or facet syndromes, spinal stenosis, lumbosacral sprain. Diagnoses may also be based on the outcome of previous treatments eg. failed back syndrome. Finally, diagnoses may be based on the history and temporal behaviour of the signs and symptoms eg. unstable low back (75). This review will focus on the anatomical structures as a source of pain, and the neurobiochemical aspects of pain as they relate to the lumbar area.

Structures producing Pain

The lumbo-sacral strain or sprain is the most common clinical diagnosis (63). Technically, the diagnosis implies injury to the muscle (strain) or ligament (sprain). The three other structures that are most frequently implicated in causing benign but significant LBP are the intervertebral disc, the facet joint and the nerve root. This paper will focus on the latter structures. The vascular system of the spine has also been implicated as aggravating in certain conditions (10). Nerve root compromise is usually a result of facet arthrosis or disc herniation. But in many cases of LBP a multiplicity of structures may be involved, especially in long term problems.

The Intervertebral Disc

Intervertebral discs have a multiple sensory supply. The lateral aspects of the intervertebral discs and the anterior longitudinal ligament receive their innervation from the grey ramus communicantes. It is not known whether this innervation is autonomic in origin (7). The posterolateral aspects of the discs

also receive innervation from this source, but in addition receive innervation from the lumbar sinuvertebral nerve. This nerve, which stems from branches of the ventral rami and grey ramus communicans supplies the posterior aspect of the disc as well as the posterior longitudinal ligament, and the blood vessels of the epidural space and vertebral bodies (39).

The receptors in the disc are unevenly distributed in the outer third of the disc. The majority of receptors are found in the lateral regions, with less in the posterior region and even less anteriorly (11). This is also the area of the disc which is most prone to injury. However, the function of the different types of receptors has not yet been determined. A vasosensory role is unlikely given the avascular nature of the disc. But a proprioceptive and nociceptive role would be reasonable (11).

Thus the disc appears to have an anatomical basis for pain if it is injured, especially in the outer areas. Provocation discographic studies have revealed that a structurally intact and myelographically normal disc will become painful if injected with normal saline. This may indicate that nociceptors respond to pressure. But, the needle will cause microtrauma in the tissues it passes through, as well as in the disc. This may elicit the release of putative inflammatory substances from the nerve terminals, thereby causing biochemically mediated pain. Also, the injected fluid can leak back along the path of the needle and irritate other structures, such as the posterior longitudinal ligament which has a rich nociceptive innervation (22). Finally, myelograms - with a false negative rate of 30% (28) are not a specific enough test to judge the normalcy of a disc. And thus some tests could have been carried out in asymptomatic but injured discs, with altered nociceptor thresholds.

Discogenic pain may be quite severe. This could be due to the multiple innervation pattern; sinuvertebral, grey ramus communicans, and dorsal primary rami to each side. It is possible that spatial summation may occur which would accentuate the nociceptive input. Diffuse nociceptive inhibitory control which would normally attenuate the nociceptive activity of the lesser input (34) would probably be a modulating factor as this input is primarily to the same spinal level.

Pain may also be severe because it may be sympathetically mediated. Autonomic innervation is present to the spine, and general autonomic effects such as sweating and nausea have been reported (23). The possibility of specific sympathetically mediated pain does not appear to have received much attention at this time, although Lipton (37) notes that there is often an element of sympathetic pain in painful back conditions after surgery.

Causalgia and sympathetic pain are probably varieties of the same condition (37). The pain has a characteristic burning sensation, often with dysthesia. The mechanisms of this type of pain relate to the sensitization of wide dynamic range (WDR) neurones in the spinal cord. These cells remain sensitised and respond to activity in large diameter A-mechanoreceptors which

are activated by light touch. This produces allodynia. Later, sympathetic efferent activity may generate A-mechanoreceptor activity and hence lead to WDR nociception (37). Other spinal mechanisms of hyperalgesia are discussed in the section on the dorsal horn.

Why does pain persist? It has been established that certain joint receptors become nociceptive after the tissue they serve has been injured (14). It has also been established that the thresholds of nociceptors decrease in the presence of inflammation (14). Also, some nociceptors show spontaneous and, or, ongoing afterdischarge activity in the presence of inflammation (14). Therefore if discogenic nociceptors react in a similar manner to nociceptors in other tissues, persistence of pain may have a neurophysiological basis.

Pain may also persist due to poor tissue healing. Bayliss et al (3) showed that a decrease in proteoglycan synthesis ocurred in spondylolisthetic L5-S1 discs. A recent study showed that stab wounds in the annulus fibrosus do not heal except for a fibrous cap which forms at the periphery (24). It is thus possible that micro trauma of the disc does not heal either. Whether this is due to the lack of vascularity of the disc, is not clear. It may be that micro trauma does not generate a strong enough inflammatory and subsequent healing response. Alternatively, the avascular nature of the disc may limit the inflammatory and the healing response. Poor quality healing leads to less tissue strength, and subsequent reinjury with minor provocation. This then sets the stage for a virtually constant state of inflammation which sensitises nociceptors, and may lead to a fairly constant state of ongoing activity. Unfortunately, accomodation does not seem to factor in to the situation.

On the other hand, Saal et al (58) suggest that they have demonstrated high levels of phospholipase A_2 in surgically excised lumbar discs. Phospholipase A_2 plays a role in the process of inflammation. However, the authors measured the specific activity levels in only five subjects and the range (291.4 to 1,014.5 nmol/min/mg) was considerable. They compared the levels of phospholipase A_2 activity in discs to other body tissue such as plasma and sperm and one has to question whether this is a reasonable comparison. In addition, no statistical analysis was offered to support their conclusions.

The question of persistence of pain may also be related to the compressive loading of the disc which occurs in any upright posture. Given that disc healing is poor, how does this loading affect the healing process?

Osti, Vernon-Roberts and Fraser (50) have shown that in sheep, even discrete peripheral annular tears tend to lead to failure and tearing of the inner annular fibres. The authors also reported that healing occured only at the periphery. These disc lesions obviously lead to changes in the mechanical properties of the disc. The changes in stiffness, viscosity and creep are analogous to that of aging (29). In addition, it seems that the size of the lesion bears no relation to these deleterious mechanical changes (29). Patterns of lumbar disc loading have been well documented (46) but whether, or how, this relates to disc healing is not clear as yet.

The compressive loading may also stimulate nociceptive activity. Time latency studies indicate that nociceptors are stimulated directly by mechanical stimuli rather than through a chemical mediator, though as mentioned earlier, the presence of certain chemical mediators such as substance P or bradykinin, will lower the nociceptive threshold (4).

The enhancement of low back pain consequent to disc herniation may also be explained by dural adhesions. Using anatomic cadavers, Parke and Wesley (51) described the disruption of neurovascular bundles containing branches of the sinuvertebral nerve, as they passed between the adherent dura and the posterior longitudinal ligament. These dural adhesions were most common in the L4-S1 region.

Another point to consider in persistent pain states, concerns the balance of activity between large fibre non-nociceptive afferent and small fibre nociceptive afferents. Although the evidence is equivocal at this point, (47) people with LBP demonstrate qualitative differences in patterns of EMG activity during movements, which implies that there are qualitative differences of movement in LBP patients compared to controls (47). Normative data on motor recruitment patterns during certain movements is a prerequisite to determining aberrations of motor recruitment in specific LBP conditions. Assuming that this is so, it is possible that this movement aberation could decrease large fibre - proprioceptive - activity. According to the Gate Control theory of pain (41) large fibre afferent activity limits the transmission of nociceptive activity. It is now known that this inhibition occurs at the spinal level, and thus there is no "gate" as such (37). Nevertheless, the basic principle is sound. And thus LBP patients may show a decrease in spinal inhibition of nociception, and therefore experience more pain and more persistent pain.

The Facet Joints

The facet joints and their associated structures are also commonly implicated in LBP. Each lower lumbar apophyseal joint is well innervated by either two (6,7,8,22,43-45) or three (74) successive spinal nerves which stem from the dorsal primary rami. The dorsal primary rami also supply the deep back muscles, and the intervertebral ligaments. Afferent fibres from joints are predominantly group III and IV, with free nerve endings forming a dense plexus in the joint capsule. The nerve endings are of four types categorised in terms of their response characteristics: 1. units excited by innocuous movement, 2. units with a monotonically increasing response to stimuli ranging from innocuous to noxious, 3. units that respond only to noxious joint movement and, 4. units which cannot be activated by any joint movement under normal conditions. However group 4 units are activated by movements within the physiological range of the joint in the presence of inflammation, once an arthritic condition is established (14).

There are conflicting views regarding nociceptive innervation of the synovial folds. Wyke (74) states that there are no receptors in the synovial

tissue or intra-articular menisci in the facet joints. On the other hand, Mooney and Robertson (45) suggest that there is a rich supply of nerves. Recent evidence has revealed neural fibres in the inferior joint recess synovial fold subsynovial tissue. Nerve fibres were found in 5 out of 13 specimens using electron microscopy, and in 8 out of 17 specimens using silver impregnation techniques (22). Giles (22) also reports that substance P immunoflurescent profiles were observed in the synovial membrane and inferior joint recess capsule. This is indicative of a nociceptive function of the receptors in this area.

The capacity of the facet joint to become painful is evident. The multisegmental innervation pattern is analagous to that of the intervertebral disc. Therefore the same factors regarding severity and persistence of pain will affect the facet joints as well as the disc. However there are some differential factors to consider in the facet joints.

The facet joints are vascular. Thus the inflammatory response will include a vascular component. Normally following tissue injury, algesic substances such as histamine are first liberated, followed by bradykinin and then prostaglandins. In addition to being released from the vasculature and other cells, these substances are liberated from mast cells which are found throughout the epineurium, perineureum and endoneurium (14). There is a dense network of neural tissue in and around the spine. Therefore, does this density affect the intensity of the inflammatory response, in terms of the liberation of algesic substances? In addition, facet joints like peripheral joints, may have type IV receptors which fire in the presence of joint inflammation. It is possible that this type IV activity contributes to the enhanced pain that accompanies joint inflammation.

The inflammatory response of the facet joints will have a greater vascular component than that of the disc. This will be positive in terms of healing but may also lead to "overhealing" as evidenced by the hypertrophic degenerative changes which occur in facet joints. This can lead to nerve root compromise due to a reduction in the dimensions of the intervertebral foramen. Also, synovitis in connection with degenerative joint disease, and dynamic impingements of synovial folds may add to the persistence or recurrance of pain from facet joints (32).

Taylor and Twomey (66) have described how the fat filled synovial folds, often become fibrous at the tips. They conclude that this is evidence of compression of these folds between the articular surfaces. However, Bogduk and Engel (9) suggest that the theory of meniscal entrapment as a cause of "acute locked back" is rather overstated.

Finally, the normal sequence of degenerative events in the three-joint complex is that one joint is affected first but then because of interplay, eventually changes will occur in all three (13). However, Butler (13) assessed facet joints using CT scans, and assessed discs using MRI. He reported that disc degeneration without facet arthritis does occur. And he suggested that

discs tend to degenerate before facet joints. Whether this relates to compressive loading, disc nutrition, proteoglycan synthesis, or other factors is not clear.

NEURAL BASIS OF PAIN

Nerve root

The "tight" anatomy of this area, means that inflammation or displacement of one structure will result in irritation of adjacent structures, thereby leading to a secondary source of nociception. The mechanical pressure and the presence of inflammatory algesic substances will lead to lowering of the nociceptor threshold and therefore nociception may result. The nerve root is at risk of becoming a secondary source of pain due to the "tightness" of spinal anatomy.

Nerve roots are structurally different in their anatomy and vascularity from peripheral nerves (65). And nerve roots do not react to compression in the same way as peripheral nerves (72). The spinal nerve root is different from a peripheral nerve in that it is encased in a flimsy gauze-like pia and has no epineurium to resist mechanical stresses. It receives its nutrition from the cerebrospinal fluid in which it is bathed, through this membrane (43). Mooney (43) suggests that it is possible that inflammation and fibrosis could obliterate this source of nutrition. He also suggests that squeezing of vessels in one area and allowing distension in another could lead to chemical imbalance (43). This chemical imbalance can lead to changes in resting membrane potential and to the generation of ectopic impulses (15). It is also possible that putative biochemicals such as bradykinin resulting from inflammation in the area, may generate nociception directly through receptors on the axon itself. If this occurs it would help to explain the severity of LBP as the area is so richly innervated.

Mechanical compression on nerve roots has also been shown to lead to blood flow compromise, which was not totally reversible (75). How much neural dysfunction is due to mechanical factors and how much is due to the secondary biochemical factors is not clear.

The answer may be complicated by such factors as rate of compression. Olmarker (48) has shown that the amount of intraneural oedema is greater with a fast rate of compression (0.05-0.1 seconds) to 50 or 200 mm Hg, compared to a slower rate (15-20 seconds) to 50 or 200 mm Hg, when applied to pig spinal nerve roots.

In a human cadaveric study, Hoyland et al (26) found that reduction of the dimensions of the intervertebral foramen was frequently associated with compression and distortion of the large venous plexus within the foramen. These authors suggest that venous obstruction may be an important pathogenic mechanism in the development of perineural and intraneural fibrosis. They also suggest that the causal link may be a result of ischaemia due to a reduced

venous outflow. However, venous outflow problems do not usually lead to ischaemic conditions.

Dorsal Root Ganglia

Spinal neural anatomy is also distinct from other areas due to the presence of dorsal root ganglia (DRG) in the intervertebral foramen. In contrast to sensory axons in nerve trunks, sensory cell somata in DRG are highly sensitive to mechanical distortion and may even discharge spontaneously (68). The incidence of spontaneous DRG discharge is significantly influenced by peripheral nerve injury (15). In 1976, Lieberman (36) wrote that the DRG were considered solely as nutritive depots for their sensory fibres, and did not play a role in signal processing. It is not known why the DRG are highly sensitive, but Devor (15) suggests that it is a function of the T-stem branching in the DRG. This branch could act as a conduction block to nerve impulses. To overcome this potential block and counterbalance the shunt, extra Na+ channels are present in the stem of the branch. These extra Na+ channels would then tend to create an impulse generating capability. And these cells would be particularly susceptible to ectopic impulse generation.

The DRG is also responsible for synthesis of the undecapeptide, substance P. Substance P is transported antidromically to the peripheral terminals where it is released. It is interesting to note that if substance P is directly introduced into the peripheral tissues all the changes associated with inflammation occur, including degradation of the mast cells (365). These authors suggest that there is a simple neuroimmunological inflammatory circuit. Thus, proinflammatory factors such as substance P are released from small diameter primary afferent nerves, and act on the mast cells to cause release of other inflammatory factors such as serotonin, histamine and leukotrienes. These factors are then responsible for the vascular dilatation and permeability of the inflammatory process, but also for the sensitisation of nociceptors. Thus the DRG may well bear the responsibility for the cutaneous pain or hyperalgesia found in radicular pain.

Investigators have looked into the question of what stimulates the antidromic transport of substance P. Mechanical stimulation of the DRG may cause ectopic impulse generation and release of substance P. Also, Rydevik et al (55) has shown that mechanical deformation causes endoneural pressure increases which led to oedema and haemorrhage in the endoneural space of the DRG. It is worth remembering that disc narrowing may actually reduce the tension on the nerve root and DRG due to a decrease in the distance that the nerve has to traverse (61).

Neuropharmocologic studies have shown that substance P in the DRG is affected by whole body vibration (73). Low frequency vibration was used as an independent variable because it is known to be a risk factor for LBP. This contrasts to high frequency vibration (80 Hz), which has a depressant effect on nociceptive neurones (73). For the former case, ten New Zealand white rabbits

were subjected to low frequency (3.5-5 Hz) vibration for two hours. Following sacrifice, DRGS were excised and subjected to radioimmunoassay for substance P levels. A control group was utilised. DRG levels of substance P were significantly lower in the vibrated rabbits. Unfortunately pre test levels of substance P are not known. And the question remains whether the vibration led to a decrease in the synthesis of substance P or, whether the vibration stimulated substance P release from the DRG which may have been transported antidromically or orthodromically to the dorsal horn.

Dorsal horn

Biochemical transportation may also be a factor in peripheral neural damage due to mechanical compression. In a controlled study, Sugimoto et al (64) reported degeneration of dorsal horn neurons in laminae I and II following constriction of the sciatic nerve in rat. This is interesting considering that orthodromic nerve impulses or chemical transportation would have to pass through the dorsal root ganglia. Whether this has any functional significance is not clear at this point.

Dorsal horn atrophy also occurs in post-herpatic neuralgia pain, but only when there is persistent pain (71). A post-mortem study of five subjects with post-herpatic neuralgia, three with persistent pain and two without pain yielded interesting differences. The three subjects with pain all exhibited atrophy of the dorsal horn at the affected level. They also exhibited cell axon and myelin loss with fibrosis in the dorsal root ganglion (71).

The discussion regarding the transmission of substance P from the DRG and the role of this substance in nociception, is complicated by the fact that substance P does not occur more frequently in nociceptive DRG cells than low-threshold mechanosensitive cells (42). At the present time the function of a DRG cell can not be predicted from its neuropeptide content and vice versa. Ju et al, cited (42) note that the interpretation of the peptide data is difficult because the concentration and combination of peptides is dependent on many factors such as hormonal influences and history of electrical activity.

In the animal model of inflammation, the superficial cells of the dorsal horn exhibit an excitability and an expansion in their receptive fields which correlates with the development of behavioural hyperalgesia (27). T
hese authors suggest that the expanded receptive fields, and the excitability of these cells is related to spinal dynorphin levels. Essentially, dynorphin and other neuropeptides promote excitotoxicity at the spinal cord level. Unchecked excitotoxicity in the spinal cord may effect small inhibitory neurones to a greater extent. This exitability shift would contribute to the observed phenomenon of receptive field expansion and behavioural hyperalgesia (27).

Neuronal plasticity of cells in the dorsal horn was demonstrated by Saito, Collins and Iwasaki (59) in the awake cat. Manipulation of spinal levels of serotonin and its antagonist methysergide was carried out. Specific neuronal activity and receptor area mapping was recorded in response to noxious

pinching. The response characteristics of the cells in the dorsal horn changed in response to the presence of serotonin or methysergide. In essence low threshold neurons developed response characteristics similar to wide dynamic neurones. The latter neurons have a nociceptive function. This is an important point to note because it provides evidence that the response profiles of neurons are not invariant (59). Pathological states in the low back may unmask these nociceptive response profiles (59) and this will contribute to the noxious input.

The complexity of neuronal and receptor activity in the dorsal horn is becoming more evident. Brandt and Livingston (12) used animal model of chronic pain (foot-rot in sheep), to test receptor activity of dorsal horn neurones thought to be involved in nociception and post-injury hypersensitivity. Saturation binding studies revealed that the number of alpha$_2$ adrenoceptors and mu opioid receptors increased. However the affinity of the receptors remained unchanged.

The results of the above study suggest that central mechanisms play a role in post-injury pain hypersensitivity. However there are still many unanswered questions in terms of the mechanisms that evoke these spinal changes, and the effect of these changes on other areas of the nervous system. The plasticity of the nervous system makes further neurophysiological changes likely. It seems that each new clue to the mystery of pain leads deeper into the enigma.

Conclusion

It is evident from the previous discussion that there is a biochemical and neural role in nociception, this is in terms of algesic substances which directly evoke activity, or which alter the threshold of the nociceptors. Endogenous substances released due to tissue damage or inflammation provide a link between noxious stimulation and nociceptor discharge (71).

In essence, a resetting of the threshold may occur so that normally non noxious mechanical or thermal stimuli may evoke activity in the nociceptors. This helps to explain the hyperalgesia which occurs with inflammation. Of interest is the fact that spontaneous activity occurs in the nociceptors after tissue damage, this activity may be implicated in persistent pain syndromes, or it may be that the thresholds do not return to their pre-injury level.

This paper discussed the mechanisms of nociception in LBP. Nociception generally plays a role in the perception of pain, but emotional and cognitive factors also contribute to the pain experience. Nociceptive mechanisms are those associated with the detection of noxious stimuli capable of compromising the integrity of the organism (4). In these terms nociceptive mechanisms are protective in nature and contribute to "normal" functional pain. But it must be emphasised that the LBP experience is much much more than nociceptive input.

REFERENCES

1. Andersson GBJ, McNeill TW. Lumbar Spine Syndromes. Evaluation and Treatment 1989:1-28.

2. Back Injuries: Costs, Causes, Cases and Prevention. Bureau of National Affairs Monograph. Washington 1988.

3. Bayliss MT, Johnstone B, O'Brien JP. Proteoglycan synthesis in the human intervertebral disc. Variation with age, region and pathology. Spine 1988:13;9;972-981.

4. Besson J-M, Chaouch A. Peripheral and spinal mechanisms of nocioception. Physiological Reviews 1987:67;186.

5. Bigos SJ, Battie MC, Spengler DM, et al. MD. A prospective study of work perceptions and psychosocial factors affecting the report of back injury. Spine 1991:16;1;1-6.

6. Bogduk N. Innervation, pain patterns, and mechanisms of pain production. In: Twomey LT, Taylor RJ, eds. Physical Therapy of the Low Back. Clinics in Physical Therapy, Vol 13. 1987;85-101.

7. Bogduk N. The innervation of the lumbar spine. Spine 1983:8;3;286-293.

8. Bogduk N. Lumbar Dorsal Ramus Syndromes. In: Grieve G. (ed.) Modern Manual Therapy of the Vertebral Column 1986:396-404.

9. Bogduk N, Engel R. The menisci of the lumbar zygapophyseal joints. A review of their anatomy and clinical significance. Spine 1984:9;5;454-460.

10. Bogduk N, Tynan W, Wilson AS. The nerve supply to the human intervertebral disc. Journal of Anat 1981:132;39-56.

11. Bogduk N, Twomey L. Clinical Anatomy of the Lumbar Spine. Churchill Livingstone, Melbourne 1987:139-147.

12. Brandt SA, Livingston A. Receptor changes in the spinal cord of sheep associated with exposure to chronic pain. Pain 1990:42;3;323-330.

13. Butler D, Trafimow JH, Andersson GBJ, McNeill TW, Huckman MS. Discs degenerate before facets. Spine 1990:15;2;111-113.

14. Campbell JN, Raja SN, Cohen RH, Manning DC, Khan AA & Meyer RA. Peripheral neural mechanisms of nocioception. In: Wall PD, Melzack R, eds. Textbook of Pain. Second edition. 1989:22-45.

15. Devor M. The pathophysiology of damaged peripheral nerves. In: Wall PD, Melzack R, (eds.) Textbook of Pain. Second edition. 1989:63-81.

16. Deyo RA, Tsui-Wu YJ. Functional disability due to back pain. A population based study indicating the importance of socioeconomic factors. Arthritis and Rheumatism 1987:30;11;1247-1253.

17. Dixon AS. Introduction. In: Jayson, MIV, ed. Third edition. The Lumbar Spine and Back Pain. 1987:xi-xii.

18. Dwyer A, Aprill C, Bogduk N. Cervical zygopophyseal joint pain patterns I: A study in normal volunteers. Spine 1990:15;6;453-457.

19. Fields HL. Core Curriculum for Professional Education in Pain. In: Fields HL (ed.) Seattle, IASP Publications. Pain Suppl 1991:60-62.

20. Frymoyer JW, Gordon SL. Research perspectives in low back pain. Report of a 1988 workshop. Spine 1989:4;12;1384-1389.

21. Gamsa A. Is emotional disturbance a precipitator or a consequence of chronic pain. Pain 1990:42;2;183-196.

22. Giles LGF. Anatomical Basis of Low Back Pain. Baltimore, Williams and Wilkins 1989:58-66.

23. Grieve GP. Common Vertebral Joint Problems. Edinburgh, Churchill Livingstone 1988:319-333.

24. Hampton D, Laros G, McCarron R, Franks D. Healing potential of the annulus fibrosus. Spine 1989:14;4;398-401.

25. Hordern A. The spectrum of stress. Stress Med 1985:1;17-25.

26. Hoyland JA, Freemont AJ, Jayson MIV. Intervertebral foramen vertebral obstruction. A cause of periradicular fibrosus? Spine 1989:14;6;558-568.

27. Hylden JLK, Nahin RL, Traub RJ, Dubner R. Effects of spinal kappa-opioid receptor agonists on the responsiveness of nociceptive superficial dorsal horn neurone. Pain 1991:44;187-193.

28. Jackson RP, Cain JE, Jacobs RR, Cooper BR, McManus GE. The neuroradiographic diagnosis of lumbar herniated nucleus pulposus: A comparison of computed tomography, myelography, CT-myelography, discography, and CT-discography. Spine 1989:14;12;1356-1361.

29. Keller TS, Holm SH, Hansson TH, Spengler DM. The dependence of intervertebral disc mechanical properties on physiologic conditions. Spine 1990:15;8;751-761.

30. Kellgren JH. The anatomical source of back pain. Rheumatology and Rehabilitation 1977:16;1;7-12.

31. Kelsey JL, White AA, Pastides H, Bisbee GE. The impact of musculo-skeletal disorders on the population in the United States. J of Bone and Joint Surg 1979:61(A);595-963.

32. Konttinen YT, Gronblad M, Korkala O, Tolvanen E, Polak JM. Immunohistochemical demonstration of subclasses of inflammatory cells and active, collegen-producing fibroblasts in the synovial plicae of lumbar facet joints. Spine 1990:15;5;387-390.

33. Kumar S, Cheng CK, Magee D. Comparison of two rake handles. In: Asfour SS (ed.) Trends in Ergonomics/Human Factors IV. Elsevier Science, North-Holland 1987:631-638.

34. LeBars D, Dickenson AH, Besson J-M. Diffuse noxious inhibitory controls (DNIC).I. Effects on dorsal horn convergent neurons in the rat. Pain 1979:6;283-30.

35. Levine JD, Coderre TJ & Basbaum AI. The peripheral nervous system and the inflammatory In: Dubner R, Gebhart GF, Bond MR (eds.) Proceedings of the Vth World Congress on Pain. Amsterdam, Elsevier. 1988:33-43.

36. Lieberman AR. Sensory ganglia. In: Landon DN (ed) The Peripheral Nerve. Chapman and Hall, London. 1976: 188-278

37. Lipton S. Pain: An update. In: Lipton S et al (eds.) Advances in Pain Research and Therapy Vol 13. New York, Raven Press Ltd. 1990:1-9.

38. Magnusson M, Granqvist M, Jonson R, et al. The loads on the lumbar spine during work at an assembly line. The risks for fatigue injuries of vertebral bodies. Spine 1990:15;8;774-779.

39. Malinsky J. The ontogenetic development of nerve terminations in the intervertebral discs of man. Acta Anat 1959:38;96-113.

40. Melzack R, Wall P. The Challenge of Pain. Second edition. London, Penguin 1988:43.

41. Melzack R, Wall PD. Pain mechanisms: A new theory. Science 1965:150;971-975.

42. Mense S. Structure function relationships in identified afferent neurones. Anat Embryol 1990:181;1-17.

43. Mooney V. Presidential address International Society for the Study of the Lumbar Spine. Where is the pain coming from. 1987:12;8;754-759.

44. Mooney V. Facet joint syndrome. In: Jayson MIV (ed.) The Lumbar Spine and Back Pain. Third edition. Edinburgh. Churchill Livingston 1987:370-382.

45. Mooney V, Robertson J. The facet syndrome. Clin Orthop 1976:115;149-156.

46. Nachemson AL. The lumbar spine. An orthopaedic challenge. Spine 1976:6;1;59-71.

47. Nouwen A, Van Akkerveeken PF, Versloot JM. Patterns of muscular activity during movement in patients with chronic low back pain. Spine 1987:12;8;777-782.

48. Olmarker K, Rydevik B, Holm S. Edema formation in spinal nerve roots induced by experimental graded compression. Spine 1989:14;6;479-573.

49. Oostdam EMM, Duivenvoorden HJ. Description of pain and the relationship with psychological factors in patients with low back pain. Pain 1987:28;3;357-364.

50. Osti OL, Vernon-Roberts B, Fraser RD. Anulus tears and intervertebral disc degeneration. An experimental study using an animal model. Spine 1990:15;8;762-767.

51. Parke WW, Watanabe R. Adhesions of the ventral dura. An adjunct source of discogenic pain? Spine 1990:15;4;300-303.

52. Pope MH, Frymoyer J, Andersson GBJ. Occupational low back pain. Praeger Press New York 1984:104.

53. Rossi U, Pernak J. Low Back Pain: The facet syndrome. In: Lipton S. et al (eds.) Advances in Pain Research and Therapy. Vol 13. New York, Raven Press Ltd. 1990:231-244.

54. Rydevik B, Brown MD, Lundborg G. Pathoanatomy and pathophysiology of nerve root compression. Spine 1984:9;1;7-15.

55. Rydevik BL, Myers RR, Powell HC. Pressure increase in the dorsal root ganglion following mechanical compression. closed compartment syndrome in nerve roots. Spine 1989:14;6;574-576.

56. Saal JA. The future of spinal medicine. In: Saal JA (ed.) Physical Medicine and Rehabilitation: State of the Art Reviews. Vol 4. No 2 Philadelphia, Hanley and Belfus Inc. 1990:379-383.

57. Saal JA. Intervertebral disc herniation: Advances in nonoperative treatment. In: JA Saal ed. Physical Medicine and Rehabilitation: State of the Art Reviews. Vol 4. No 2. Philadelphia, Hanley and Belfus Inc. 1990:175-190.

58. Saal JS, Franson RC, Dobrow R, Saal JA, White AH, Goldthwaite N. High levels of inflammatory phospholipase A_2 activity in lumbar disc herniations. Spine 1990:15;7;674-678.

59. Saito Y, Collins JG, Iwasaki H. Tonic 5-HT modulation of spinal dorsal horn neuron activity evoked by both noxious and non-noxious stimuli: a source of neuronal plasticity. Pain 1991:40;205-219.

60. Salter MW, Henry JL. Differential responses of nocioceptive vs. non-nocioceptive spinal dorsal horn neurones to cutaneously applied vibration in the cat. Pain 1990:40;311-322.

61. Spencer DL, Miller JAA, Bertolini JE. The effect of intervertebral disc space narrowing on the contact force between the nerve root and a simulated disc protusion. Spine 1984:9;422-426.

62. Spengler DM, Bigos SJ, Martin NA, Zeh J, Fisher L, Nacnemson A. Back injuries an industry: A retrospective study. I. Overview and cost analysis. Spine 1986:(11);241-245.

63. Statistics Canada. Work Injuries 1985-1987. Supply and Services, Ottawa. 1988.

64. Sugimoto T, Bennett GJ, Kajander KC. Transynaptic degeneration in the superficial dorsal horn after sciatic nerve injury: effects of a chronic constriction injury, transection and strychnine. Pain 1990:42;2;205-214.

65. Sunderland S. Traumatised nerves, roots and ganglia: Musculoskeletal factors and neuropathological consequences. In: Korr M (ed.) The Neurobiologic Mechanisms in Manipulative Therapy. New York, Plenum Press 1978:137-166.

66. Taylor JR, Twomey LT. Age changes in lumbar zygopophyseal joints. Observations on structure and function. Spine 1986:11;1986;739-745.

67. Tollison CD. Assessment and treatment at Pain Therapy Centers programs. In: Tollison CD. Handbook of Chronic Pain Management. Baltimore, Williams and Wilkins, 1989:656-663.

68. Wall PD, Devor M. Sensory afferent impulses originate from dorsal root ganglia as well as from the periphery in normal and nerve injured rats. Pain 1983:17; 321-339.

69. Watson J.Pain and nocioception - mechanisms and modulation. Modern Manual Therapy of the Vertebral Column. Grieve G. (ed.) Edinburgh, Churchill Livingston, 1986:206-232.

70. Watson CPN, Deck JH, Morshead C, Van der Kooy D, Evans RJ. Post-herpatic neuralgia: further post-mortem studies of cases with and without pain. Pain 1991:44;105-117.

71. Weinstein JN. Recent advances in the neurophysiology of pain. In: Saal JA (ed.) Physical Medicine and Rehabilitation: State of the Art Reviews. Vol 4. No 2. Philadelphia, Hanley and Belfus Inc. 1990:201-219.

72. Weinstein J, Pope M, Schmidt R, Seroussi R. Neuropharmocologic effects of vibration on the dorsal root ganglion. An animal model. Spine 1988:13;5;521-525.

73. Wyke BD. The neurology of low back pain. In: Jayson MIV (ed.) The Lumbar Spine and Back Pain. Second edition. Kent Pitman Medical 1980:265-339.

74. Yoshizawa H, Kobayashi S, Kuboto K. Effects of compression on intraradicular blood flow in dogs. Spine 1989:14;11;1220-1225.

CHAPTER THREE

PSYCHO-SOCIO-MEDICAL BASES OF LOW BACK PAIN[2]

Introduction

Pain is as elemental as fire or ice (66) However, unlike fire or ice it remains an enigma. In its chronic pathologic form, pain exacts severe emotional and physical stresses on the patient and on their family. Most episodes of low back pain (LBP) are benign and resolve spontaneously (83). Unfortunately a growing number of people are disabled by benign LBP. Pain and disability pose one of the most challenging interdisciplinary research and clinical problems.

The prevalence rate of LBP is estimated at 80% (9). Thirty million individuals are afflicted with chronic low back problems in the United States (2). It is estimated that 50,000,000 chiropractic and 5,259,000 physical therapist visits are made for LBP each year (71).

The rate of disability due to LBP is more variable. In the United States 9,200,000 are currently impaired by low back pain but 2,400,000 are disabled. The estimated rate of disability due to back pain is 0.4% of the population. This compares to a disability rate of 0.158% in Britain (45), supporting the notion that disability is related to psychosocial factors(33).

It is disconcerting that as the knowledge of pain and disability has increased so has the rate of pain related disability. Disability due to LBP has increased to epidemic proportions in Western society (67). This is particularly troublesome because it is present in relatively young people i.e. less than 45 years of age (44). Thus low back pain frequently results in time loss from work.

In Quebec in 1987, wage loss replacement costs for 1981 were $129 million, whilst medical costs amounted to $21 million. Wage replacement tended to account for 86% of the costs per claim. It was further reported that 7.4% of the claims accounted for 75% of all workers compensation costs (80). This imbalance of costs per claim is a robust phenomenon reported by others (24,92,93).

These figures reflect the true magnitude of the problem of LBP related disability. It is the disability that is the problem, not the LBP. This is true in financial terms and in terms of physical limitation, psychological distress and suffering. Low back pain itself, is so common that it should be regarded more as a normal part of life than as a medical problem that requires treatment.

Chronic LBP is associated with physical, psychological and social problems. The most frequent psychological problems include depression,

---

[2]A version of this chapter has been submitted for publication. Simmonds, Kumar and Lechelt 1993. Disability and Rehabilitation

anxiety and social isolation (26,66). These psychological and sociological difficulties are especially marked for patients with pain and symptomatology that is incongruent with physical pathology (54,79,108,111). It has been suggested that the treatment of chronic pain is influenced to a greater extent by the patient's distress and demands for help and to a lesser extent by the severity of the physical disease (108). Unfortunately in lay usage and clinical practice, pain is regarded primarily as a sensory symptom of a physical disease (109) and is treated accordingly. This leads to iatrogenic induced disability, and to feelings of frustration, anger and helplessness on the part of both patient and physician (102).

It is uncertain why some patients enter a spiral of inactivity and distress while others manage to maintain almost normal function (75). Early psychologically based attempts at discriminating between the "enabled" and disabled used characteristics such as; personality profile (21,96,113), coping strategies (82,106), and presence of depression (17). Socioeconomic constructs such as litigation and employment status (98,107) "hassles" (16), spousal influence (39,55), pain behaviour (42), symptom and illness behaviour (74,110) have also been explored. The patient, their back, their personality, and their social milieu have been examined in depth, in an effort to discriminate between the many with LBP but minimal dysfunction and the few who progress to disability.

Much less research has focussed on the role of the medico-legal-social system itself, and on the clinicians, lawyers and claims adjudicators working within this system. But it is physicians who provide the letters of support for disability entitlements, the prescriptions for medication, and who order and arrange the multiplicity of investigations and interventions. Lawyers working on a contingency fee basis may encourage frivolous suits or undermine return to work (60). The lack of empirical knowledge in this area is acknowledged (3,8,72). But, recent attempts have been made to address the role of health professionals in pain related disability (75).

In sum, pain and pain related disability are complex questions and a complex answer seems to be emerging. Single factors or unidimensional constructs are predictably too simplistic to be helpful. A great deal of research has focussed on discriminating between the many with LBP but minimal dysfunction, and the few with LBP that progress to disability. Given the complexity of the problem it seems intuitive that discriminability for level of disability, and prediction of outcome will be optimal if it is based on a multifactorial model with physical, psychological and sociological dimensions.

The purpose of this paper is to review psychosocial factors that play a role in the disability associated with LBP. The contributory role of anatomical and pathophysiological factors in LBP has been previously reviewed (91).

Psychological predictors of Low Back Pain

The "pain-prone masked depressive"?

The main reason why disabling back pain remains enigmatic is because the focus has been too narrow, in studying (107), preventing (6), assessing (35), and in treating LBP (44).

Traditionally the medical model focussed treatment solely on the physiological domain. Patients who failed to respond to this treatment were dismissed as having psychogenic pain. Patients with LBP have, as a group, been maligned. Terms such as "functional overlay" and "psychogenic back pain" have been accepted as self evident truths by many clinicians. But they are misrepresentative and misleading terms "conjured to shroud ignorance" (29) and perhaps to displace blame. It is disconcerting that these terms are used and accepted given the acknowledged inadequacies of present diagnosis in the majority of LBP cases.

The origin of the term "psychogenic pain" is credited to George Engel (26,87). In 1959 Engel presented a theoretical report concerning patients with idiopathic intractable pain. He labelled the pain psychogenic, and the patients pain-prone. Using a psychodynamic interpretation, he characterised the personality profile of these patients (21). In an extension of this personality model, Blumer and Heilbronn (7) suggested that chronic pain reflects a muted or masked depressive state in a pain-prone individual. The main clinical features of the pain-prone personality include: 1. somatic complaints (continuous pain of obscure origin, hypochondriasis, desire for surgery), 2. "solid citizen" mentality (denial of conflicts, idealization of self and family relations), 3. depression (fatigue, inactivity, inability to enjoy social life), 4. history (family or personal history of depression and alcoholism, past abuse of spouse). In summary, Blumer and Heilbronn (7) argue that the pain is a conversion of unbearable guilt and anguish that is repressed and displaced onto the body in pain-prone individuals.

This simplistic psychodynamic view of pain has been increasingly questioned and the original research has been seriously challenged on conceptual, methodologic and statistical grounds (23,26,30,32,64,70,81,85,86,88,90,100,105,115). In sum there appears to be little evidence to support this psychodynamic view of a pain-prone personality.

Depression

In support of the notion of a psychologically homogenous group, Blumer and Heilbronn (7) argue that many depressed psychiatric patients report pain symptoms. However, the comparison between psychiatric and chronic pain patients does not hold (104). The rate of depression in chronic pain patients has been reported to be as high as 100% and as low as 10% (104).

Patients with chronic pain of musculoskeletal origin do have higher rates of depression than pain free subjects, 18% and 8% respectively (57). However,

it is reasonable to attribute the depressed mood to the frustrations of having a pain problem that is not understood by health care professionals (87) and which limits activity. This view is supported by Atkinson et al (4) who report that depressed mood appeared to be a direct consequence of back-pain related life events.

The discrepancy in the prevalence rate of depression (10-100%) can be attributed to the assessment bases used and the population tested. In an examination of the Hispanic Health and Nutrition Survey data, Magni et al (57) reported that the rate of depression amongst Mexican and Cuban Americans with abdominal pain was 18.7% or 6.8% depending on the scale used. The Depression Scale of the Center for Epidemiologic Studies gave far higher rates of depression than the more conservative Diagnostic Interview Scale.

Many reports on the prevalence rates of depression have been based on patients referred to specialty pain clinics. The difficulty here is that these patients are not representative of the pain population in general and tend to have more psychological difficulties (26,104,108). Many have been referred to pain clinics because they are "failures" of the traditional healthcare system (104). However, there is increasing evidence that it is the healthcare system that has "failed" the patient. Of 89 patients referred to an in-patient behavioral pain management unit, only 3 patients were found to have had no inappropriate treatment, prescription, investigation, referral, advice or explanation (75).

Cause or Consequence

Evidence for the argument that severe or persisting emotional distress can trigger new pain or reinstate old pain in the absence of pathology does not extend beyond clinicians' reports (13).

Gamsa (26) explored the question of whether emotional disturbance is a cause or consequence of chronic pain. She found that chronic pain was consistently associated with current emotional disturbances, manifested as depression and diminished life satisfaction. However, the author reported that there was little relationship between pain and preceding events such as unmet childhood needs or parental overprotection. This argues against the predisposition of a person to pain and dysfunction, based exclusively on emotional factors.

"Adult difficulties" such as spouse abuse, drug or alcohol abuse and relatives suffering from chronic pain or crippling disorders were found to be significant. However, one would be hard pressed not to become depressed with such life events, with or without LBP. Overall Gamsa suggests that emotional disturbances were consequences of chronic pain rather than precipitators (26).

Polatin et al (77) made the somewhat exaggerated claim to be first to differentiate psychological causes vs psychological consequences. These findings are not in total agreement with those of Gamsa (26). However, they

may be explained in terms of differences in the population, in the psychological disorder and in the investigative methodology.

Polatin and colleagues (77) assessed 200 chronic LBP patients entering a functional restoration programme, whereas Gamsa's (27) subjects were recruited from a general practitioner, a physical therapy clinic, and a pain clinic. Polatin used a structured psychiatric interview and reported that their patients had high rates of psychiatric diagnoses (55%). The most common diagnoses were depression, substance abuse, and anxiety disorders. Of interest, was the fact that 94% of patients with substance abuse and 95% of those with anxiety disorders had experienced these syndromes before the onset of their chronic back pain. Depression illustrated a different pattern. Depression preceded the problem in 54% and was a consequence of LBP in 46% of the patients (77).

These findings suggest that psychiatric disorders such as anxiety and substance abuse are risk factors whereas depression is more likely to be a consequence of pain. The high rate of psychiatric disorders should be viewed with caution. All patients were referred to a specialised facility and had back problems for more than three months. This indicates that some difficulty with resolution of symptoms had been identified, and the patients were in the high risk time range category for not returning to work (78).

Two other points are important in the study by Polatin. First, the diagnosis of somatoform pain disorder is recognised as being subject to individual interpretation (77). Second, backache is extremely common but the pathophysiology is usually obscure (17). The lack of a specific organic diagnosis biases the diagnosis of somatoform disorder.

In another study Gamsa and Vikis-Freibergs (25) examined the role of psychological events as risk factors or consequences. They measured the specific traits identified by Blumer and Heilbronn (7) that characterize the pain-prone individual. A total of 244 subjects were tested, 163 with chronic pain and 81 controls. Of the 20 measures employed, only general emotional repression and ergomania were significantly different between groups. In addition, individuals in the pain group were more likely to have a relative with pain. Contrary to the proposition that patients with pain are emotionally repressed and pain is a somatic expression of these feelings, the pain group was much more likely to express their pain related emotions. Moreover, the more pain they had the more likely they were to express their feelings (25). The authors interpret the relationship between emotional expression and pain in terms that these patients are suffering emotional difficulties because of their pain, and their greater emotionality is their normal response in any situation (25). This greater emotional reaction to the pain may however increase their pain complaints and perceived distress leading to overtreatment on the part of the health practitioner.

The increased levels of ergomania (defined as beginning to work at an early age, frequent overtime and infrequent vacations) in the pain group, may

be a psychodynamic problem. However it may also be explained in biomechanical terms. Physical stress leads to physical "wear and tear" and is recognised as a risk factor for back problems (11,49,53,120). Further, work habits or "ergomania" cannot be judged in a vacuum. Social, vocational and economic pressures also contribute to work habits. Finally, it may also be the case that whether a person is judged negatively as an "ergomaniac" or positively as having a strong "work ethic" may depend on who is the judge.

As noted earlier, there is little support for the pain-prone personality. A recent report by Wade et al (112) supports this position and reports that most people with chronic pain have a normal personality structure. Wade analyzed a group of 88 patients referred to a multidisciplinary pain programme. Of these 88 patients, 59 met the criteria for group classification using the Minnesota Multiphasic Personality Inventory (MMPI) (96). Only the emotionally overwhelmed group, of which there were only 12 had high neuroticism scores, and could be differentiated from the other patients in terms of higher levels of depression, anxiety, vulnerability and hostility. All the other patients had scores on the MMPI within the normal range. Moreover, feelings of depression, anxiety, vulnerability could easily be attributable to inappropriate cognitions about the back problem and the potential effect of this problem on employment, family and social life. The results from this study support the position that patients with pain problems are psychologically normal. But they do have a problem in managing their pain, and they may worry and get depressed about the impact of the back pain on their lives.

Families and pain

Gamsa and Vikis-Freibergs (25) found that patients with pain are more likely to have a relative with chronic pain. This finding is consistent with the Nuprin Pain Report (97). Explanations for this finding have been based on the theory of social modelling (12), genetic vulnerability, or common environment stressors (25). However, it may also be explained in much simpler terms. Given a prevalence rate of 80% LBP it is highly likely that a family member will have pain by chance alone.

Edwards et al (20) examined the relationship between family history of pain and current pain experience. Two hundred and eighty eight college students participated in the study. The participants completed a pain questionnaire on current personal pain symptoms, and whether other family members had persistent pain. The participants reported a mean of six pain models in their families. Given that the participant group was a college group not selected on the basis of pain, these results argue against the usefulness of "presence of pain in family members" as a predictor variable for pain. However, the effects of "presence of pain in family members" potentially provides much more useful information.

It is reasonable to assume that chronic pain or chronic illness will have cognitive, affective, behavioral, social, economic and other consequences for

the individual and the family. Chronic pain can be perceived as a crisis for the family. But a crisis can be a threat or a challenge (103). Families, like LBP patients are not homogenous. How the family adapts will depend on the state of the family when the problem occurs, and how the problem affects their relationships (103).

Social support is generally associated with better adaptation, and it is also associated with pain behaviour (28). Gil and colleagues found that pain behaviours such as guarding, rubbing and bracing, varied as a function of the satisfaction with social support rather than the number of support persons (28). A factor of quality rather than quantity!

Flor, Turk and Rudy (23) examined the role of significant others (SO) in reinforcing pain behaviours. Participants in this study were 84 male and 101 female chronic pain patients referred to a pain management clinic. The patients and their SO completed the Multidimensional Pain Inventory (MPI) and SO-MPI. This inventory assesses pain intensity, and the interference of the pain on different areas of life, mood, life control and spousal support. The results of this correlational study suggest that there is some support for the notion that SO's play a role in reinforcing pain behaviours. However the extent of this reinforcement is dependent on gender, marital status and marital satisfaction. In essence, the relationship between pain impact and SO reinforcement behaviour e.g. solicitous behaviour, was strongest between male patients and SOs who were satisfied in the marital relationship.

Wives seem to be more affected by their husbands' pain, than husbands are about their wives' pain. This finding is in agreement with the study by Rowat and Knafl (84). These authors reported that there were more distressed females than distressed males. Factors of uncertainty and helplessness were central to this distress. It was also found that highly distressed spouses tended to rate the patient's pain at a higher intensity than the patient did himself. The converse was true for low distress spouses. In addition, the low distress couples were more knowledgable about factors which impacted on the patient's pain. Thus, they would have a sense of control over the pain and be less likely to feel helpless. Finally, the low distress couples suggested that the pain had brought them closer together (84). As noted earlier pain can be perceived as a threat or a challenge. A challenge that is met and overcome is a catalyst for personal growth.

Another finding reported in this study was that 83% of the spouses reported experiencing some form of health problem which they attributed directly to living with partners in chronic pain. Sixty nine percent attributed their health problem to emotional stress. Twenty three percent described physical symptoms and 8% described social disruption eg., feeling house bound.

Locus of control and coping strategies

Psychosocial factors are particularly relevant in the area of coping and in the development of coping strategies in response to chronic pain. Recent

research has suggested that efficacy of these coping strategies are associated with adjustment in chronic pain patients and in their families (1,15,39,40,41,51). Patients who believe that they can control their pain, avoid catastrophyzing about their condition, and also believe that they are not severely disabled appear to function better than those who do not (40).

Coping strategies are associated with health and pain attributions and with self-efficacy beliefs. The impact of disease on an individual, as well as response to treatment is related to psychological characteristics (118). A psychological construct, that is relevant to pain and disability, is that of the Multidimensional Health Locus of Control (MHLC) (114). This scale consists of three subscales: internal health locus of control (I-LOC), powerful others health locus of control (P-LOC) and chance health locus of control (C-LOC). Patients with a high I-LOC have a greater belief in their own capacity to cope with, or to reduce pain. Patients with a strong belief in P-LOC mainly rely on health professionals to reduce their pain. Finally, patients with a C-LOC believe that their health situation depends on luck or fate (14).

There is an intuitive notion that a positive response to treatment would correlate with a P-LOC and there is investigative support for this (68). However, there is also evidence that supports a strong correlation between I-LOC and good treatment outcome (34). Recent evidence has shown that the best predictor of perceived pain is LOC style (5). Subjects with an I-LOC perceive less pain. This helps to explain why these patients have less psychological distress and probably less disability. It also explains why the patients tend to have a positive response to traditional treatment. Low levels of perceived pain intensity are much easier to control through conventional means. Furthermore, successful control of pain will strengthen self-efficacy beliefs which are associated with successful coping strategies in clinical pain (58).

Coping, is itself not a unidimensional construct. Moreover, specific coping strategies must be dynamic if they are to be deemed appropriate in a given situation. The appropriateness of specific coping strategies varies with the population and with pain intensity.

For instance, in a chronic pain population, "attentional" coping strategies (information seeking and viewing circumstances more favourably) was associated with less depression and anxiety than avoidant strategies (e.g. eating more), whilst in an acute pain population "avoidant" strategies were associated with less depression, anxiety and more activity (36).

In a rheumatoid arthritis population, pain severity influenced coping strategies and positive mood (1). The use of distraction and seeking emotional support was related to positive mood if the patient had a low level of pain intensity. The converse was true in patients with high levels of pain, fewer of these activities were associated with positive mood. The authors of this study also reported that subjects who exhibited more coping strategies were more likely to exhibit less pain and more positive mood over the course of the study (75 days). This makes intuitive sense given that different strategies are

differentially appropriate. The decline in pain levels may also be supportive of the adaptation effect of chronic pain (80). Further it supports the notion that successful coping improves the belief in self efficacy.

Keefe et al (41) analyzed coping strategies in a low back pain population. Patients scoring high on the "helplessness" scale of the Coping Strategies Questionnaire tended to have more psychological distress. However coping scores were not related to activity measures. Thus coping seems to be important in relation to the psychological adjustment to pain but is less useful in relation to the determination of physical ability. It seems that psychological dysfunction is more closely related to physical limitation than it is to pain (69). This again supports the focus of functional restoration programmes.

Further confirmation of this stems from other work of Naliboff et al (69). MMPI profiles were compared between a group of chronic pain patients and a group with diabetes in which there was no pain, but there was restriction of activity (70). Similar profiles were reported between both groups indicating that restriction of activity by pain or by illness evoke similar psychological changes. These changes are usually reflected by higher levels on the hypochondriasis and depression scales, and this makes intuitive sense in both patient groups. In addition, psychological adjustments may occur secondly to physical restriction, but revert to "normal" following relief of symptoms (95).

The importance of the association between physical restriction and distress rather than pain perception and distress, supports the current trend away from the prescription of rest and towards activity. This supports the value of functional restoration programmes e.g. (59).

Further support for physical activity was reported by Gatchel et al (27). These authors showed that psychologic measures paralleled improvement in physical function. They used a battery of psychological tests including the MMPI, Beck Depression Inventory, and a Quantitated pain drawing, to measure psychological adjustment.

In a later study comparing patients for whom the programme was successful compared to those for whom it was not, the level of pain was a significant predictor (76). Those with higher levels of pain and disability did not tend to benefit from the programme. In addition those with a job or with more seniority tended to obtain more benefit from the programme. However, these are the same factors that predict return to work anyway (76). This argues for the need for a controlled randomised trial in order to test the specific benefits of the programme.

The key point is that a number of factors were found to predict success and these were physical-medical and psychosocial. This supports the complexity of the pain disability conundrum, and provides further proof that neither the medical model nor the psychodynamic model of pain and disability are adequate alone.

It should be recognised that the results must be interpreted with caution as there was no control group in Polatin's (76) study. In addition, it is quite

possible that the study population may have been biased in terms of their level of psychological distress. The subjects were referred to a specialty spinal clinic because they were "failures of conventional/surgical care". This statement clearly places the blame for failure on the individual patient. A more appropriate statement may be that "conventional/surgical care had failed the patient"! The iatrogenic role in disability must be acknowledged, and accepted if the rate of disability is to decrease.

Socioeconomic and Iatrogenic Factors in Disability

Socioeconomic factors have long been recognised as influential factors in pain and disability. Traditionally it has been the patient, his complaints, behaviours and motivations that have been scrutinised. However there is a shift of focus onto the role of health professionals, lawyers and insurance bureaucrats and the contribution that they make to the problem of disability (8,60,72,75).

Pain and disability have become a big business. There has been a virtual explosion in the number of centres or programmes focused in whole or part towards the management of chronic pain and disability, moreover the motivation of this growth industry has been questioned (3,8).

Bonica (8) has stated that some pain clinics are run by unscrupulous physicians and non-physicians using the current state of interest to exploit patients. He uses the example of the surge of acupuncturists in the 1970's when public interest in the treatment was at its height, to make his point.

In the United States, less than 5% of these clinics are accredited by either the Joint Commission on Accreditation of Health Care Organizations or the Commission on Accreditation of Rehabilitation Facilities (94).

Tearnan and Cleeland (102) conducted a survey of the attitudes of physicians toward chronic pain patients. Using a ten point scale of agreement (10 = agreement, 1 = disagreement), physicians were asked whether they agreed or disagreed with the statement that, chronic pain was often iatrogenic. Psychiatrists and internal medicine specialists tended to agree (average score = 6.3). Oncologists did not have the same view (average score = 2.5). This is probably a reflection of the patient groups that they deal with. As a group physicians did not strongly endorse items reflecting adverse attitudes and misconceptions about chronic pain patients (102). This may be true, but the results should be interpreted with caution. The response rate to the survey was only 56%. So the sample may be biased. Moreover there was a differential response rate amongst physician groups, 75% for neurologists, compared to 45% for surgeons. Given the differences in levels of agreement between specialists this may influence the outcome so that the grouped scores regress to the mean. In addition, the responses given may be a reflection of the perceived socially correct response, rather than the true personal response.

There is presently a great deal of interest in accountability and cost-effectiveness of all medical treatment including pain related disability (94).

The issue of efficacy and accountability should be seen as a challenge not a threat to the socio-medico-legal system!

Financial issues have always played an important role in pain and injury and there is a body of literature dealing with pain, disability, compensation and litigation e.g. (60,116,117). Disability is a legal term and the extent of disability is based on whether a person can engage in gainful activity (119). It is based on sociopolitical factors as well as medical impairment. Thus disability judgements vary from country to country and from agency to agency (72). In general terms disability and financial recompense are directly related. Hence the assumption that financial secondary gain acts as a disincentive to ability and "rewards" disability.

Self-evident truths based on simplistic models do not hold for the prediction of pain nor do they for the prediction of disability.

Compensation, Pain and Disability

The self-evident truth that secondary gain leads to malingering, "accident neurosis" (65) or "compensation neurosis" (46) is not upheld in fact. Rather the effects of compensation on chronic pain and illness behaviour is mediated by a complex interaction of biological, (53) physical (99) psychological (31,63) and social factors. It is also mediated by economic factors, vocational factors (20,54,100) and the legal and adversarial characteristics of the particular compensation system (10,37,62,98).

The complexity of the factors involved, and differences in methodology explain the inconsistency of findings between studies. The confusion in the literature may have served only to enhance the biases of the reader (73).

Krusen and Ford (48) examined the medical records of 509 patients with low back pain for differences that may have been related to compensation. Fifty-five percent of patients on compensation (WCB) were rated as improved compared to 88.5% of non-compensation patients (NWCB). Patients were classified as "unimproved" if they continued to complain and failed to resume normal activities including work. Major procedural concerns are associated with this study which make the inferences drawn suspect. These include problems such as sampling bias, heterogeneity between subject groups, and the insensitivity and subjectivity of outcome.

More specifically there were many differences between the groups apart from compensation status. First, 22% of WCB had symptoms for more than three months before treatment, whereas only 7% of NWCB had waited that long. As expected there was a negative association between duration of symptoms before treatment and outcome. Secondly, differences in the number of treatments between groups were noted. The WCB group had a mean of 18 treatments compared to the NWCB who had a mean of 10 treatments. Further, the authors note that the treatments may not have been adequate. They suggest that no treatment is better than inadequate treatment, and certainly it should not be continued for prolonged periods of time. The authors suggest

that compensation patients as a group present different psychological problems than those not receiving compensation. They also suggest that once the cash settlement is made, the back pain and disability subside rapidly (115). Yet they present no evidence to support either of these claims. Several other studies found no significant differences related to compensation status; in pain severity, psychological distress, or attitudes and coping styles towards medical treatment (50,52,61,63).

Mendelson (63) compared 47 WCB patients with LBP and 33 NWCB patients for pain complaints and psychological disturbance using a battery of tests. He found no difference between groups on ratings of pain severity, or pain description and no difference between groups on psychological disturbance. He did however report that both pain groups had high levels of depression and anxiety compared to the normal population (63).

Leavitt et al (52) examined organic status, psychological disturbance and pain report characteristics in patients admitted to hospital for the evaluation of LBP. The volunteers were assigned to one of two groups which differed only on WCB status. Leavitt reported that the only difference between the two groups was that the WCB group described their pain using more words. Moreover, this finding was only present in the WCB group that had distinct organic signs of LBP and were not psychologically disturbed (52).

Finally, Labbe et al (50) used the Millon Behavioral Health Inventory in order to determine whether WCB and NWCB patients differed in their attitudes towards and coping styles towards medical treatment and in their interactions with medical professionals. The study sample of 283 chronic pain patients were drawn from a comprehensive pain and rehabilitation centre. No differences were found between WCB and NWCB patients on all coping styles and psychogenic attitude scales (50).

The results from controlled trials don't lend support to the concept of compensation neurosis or psychological problems. This seems to be a robust phenomenon that holds in different study populations (general hospital and specialised pain centre), and with the use of different measures for pain, psychological problems and pain behaviour.

However, none of the studies considered the potential confounding effect of employment status, or litigation vs. compensation status. Dworkin et al (19) examined the effects of compensation, litigation, employment, and short and long term outcome in a total of 454 chronic pain patients. They found that compensation and employment status predicted a poorer short term outcome, but only employment status predicted long term outcome.

Furthermore, this issue may be confounded, because patients who were employed were better educated. This suggests that they may have been involved in less physical work, and therefore be able to resume work earlier. Leavitt (53) investigated this question and found that the amount of physical exertion involved in a job does prolong recovery time. But the effect of injury on the job is to prolong recovery further (53).

It is possible that there is a differential effect of compensation. WCB has no significance on the psychological domain, but may have an effect on the physical domain. This issue is addressed in a study by Javid (38). Javid examined the effect of compensation on the signs and symptoms following chemonucleolysis. The study can be critiqued because it was not blinded. However chemonucleolysis was performed in 214 patients, follow up examinations were conducted at six weeks and six months and a questionnaire follow up was conducted at one year. The one year follow up results showed that chemonucleolysis was successful (judged as subjective pain relief) for 60% of WCB compared to 91% of NWCB. The six week and six month evaluations showed similar trends and tend to support the argument that the presence of compensation payments influences treatment outcome. There was no difference between groups for the presence of sciatica, paraesthesia, forward flexion, tenderness, sensory loss and reflex changes at six weeks. However, at six months the situation changed and differences were noted between groups on all measures except tenderness and reflex changes. The author provides a great deal of detail about the chemonucleolysis procedure but does not provide an interpretation of the results, especially in regards to the different pattern of results over time.

Why do WCB and NWCB diverge with time, based on physical signs and symptoms? The compensation system itself may be a major source of stress (31). Uncertainty about the outcome of litigation could increase stress and disability. In a comparison of compensation systems, Carron et al (10) reported that only 12% of patients within the United States compensation system had returned to full activity, compared with 27% of recipients in the New Zealand compensation system. Moreover, patients in the United States system used more medication, were more restricted socially and were more irritable. The authors attribute these differences to the adversarial relationship in the United States system, and the study by Javid (38) was conducted in Wisconsin.

Summary

There is little evidence that supports pain prone personality profiles as predictors of low back pain. However there is strong evidence that supports the role of psychosocial factors in predicting ability or disability subsequent to low back pain. Pain is associated with depression but the depression is secondary to the restriction of function. The ability to cope as an individual and as a family are also important factors in predicting ability. Finally, the medico-socio-legal system must take responsibility for contributing to the problem rather than the solution.

Physical deconditioning and adversarial relationships in the medico-socio-legal system can increase the physical problems associated LBP. The reduction of physical capacity can lead to depression and to anxiety about the future. This may be manifested as distress. This downward spiral continues. Distress results in a decrease in physical functioning and an increase in bodily

awareness. These symptoms present clinically as inappropriate responses to physical examination (111). In turn this leads to "inappropriate treatment behaviour" (75) unnecessary investigations and treatments which contributes to the financial and human costs associated with pain related disability.

LBP related disability may be manifested by the individual, but it is a result of social as well as individual factors. The problem of LBP related disability is unlikely to diminish unless the aggravating factors in the medico-socio-legal system are addressed. The economic crisis in health care costs may provide the social and political will to address those factors in the medico-socio-legal system which contribute to the problem, rather than the solution of LBP related disability.

REFERENCES

1. Affleck G, Urrows S, Tennen H, & Higgins P. Daily coping with pain from rheumatoid arthritis: patterns and correlates. Pain 1992:51;221-229.

2. American Medical Association. Guides to the Evaluation of Permanent Impairment. 3rd edition revised. Milwaukee, American Medical Association, 1990.

3. Aronoff GM, McAlary PW, Witkower A & Berdall MS. In: Aronoff GM (ed) Pain Centers: A Revolution in Health Care. New York: Raven Press, 1988:123-136.

4. Atkinson JH, Slater MA, Grant I, Patterson TL, & Garfin SR. Depressed mood in chronic low back pain: relationship with stressful life events. Pain 1988:35;47-55.

5. Bates MS, Edwards WT, Anderson KO. Ethnocultural influences on variation in chronic pain perception. Pain 1993:52;101-112.

6. Bigos SJ, Battie MC, Spengler DM, et al. A prospective study of work perceptions and psychosocial factors affecting the report of back injury. Spine 1991:16;1,1-6.

7. Blumer D, Heilbronn M. Chronic pain as a variant of depressive disease: the pain-prone disorder. Journal of Mental and Nervous Disease 1982:170; 381-406.

8. Bonica JJ. Evolution of multidissciplinary/interdisciplinary pain programs. In: Aronoff GM (ed) Pain Centers: A Revolution in Health Care. New York: Raven Press 1988:9-32.

9. Bureau of National Affairs Monograph. Back Injuries: Costs, Causes, Cases and Prevention; Washington, Bureau of National Affairs, 1988.

10. Carron H, DeGood DE, & Tait R. A comparison of low back pain patients in the United States and New Zealand: Psychosocial and economic factors affecting severity of disability. Pain 1985:21;77-89.

11. Clemmar DI, Mohr DL, Mercer DJ. Low back injuries in a heavy industry I. Worker and workplace factors. Spine 1991:16;7;824-830.

12. Craig KD. Social modelling influences: pain in context. In: Sternbach RA (ed) The Psychology of Pain (2nd ed) New York: Raven Press, 1978:67-96.

13. Craig, K. Emotional aspects of pain. In: Wall PD and Melzack R.(eds). Textbook of Pain (2nd edition), Churchill Livingston, Edinburgh, 1989:220-230.

14. Crisson JE, Keefe FJ. The relationship of locus of control in pain coping strategies and psychological distress in chronic pain patients. Pain 1988:35;147-154.

15. Crook J, Tunks E, Kalaher S, & Roberts J. Coping with persistent pain: a comparison of persistent pain sufferers in a specialty pain clinic and in a family practice clinic. Pain 1988:34;175-184.

16. De Benedittis G, & Lorenzetti A. The role of stressful life events in the persistence of primary headache: major events vs. daily hassles. Pain 1992:51;35-42.

17. Deyo RA. The role of the primary care physician in reducing work absenteeism and costs due to back pain. In: Spine: State of the Art Reviews 1987:2;1,17-30.

18. Doan BD, Wadden NP. Relationships between depressive symptoms and descriptions of chronic pain. Pain 1989:36:75-84.

19. Dworkin RH, Handlin DS, Richlin DM, Brand L, & Vannucci C. Unraveling the effects of compensation, litigation, and employment on treatment response in chronic pain. Pain 1985:23;49-59.

20. Edwards PW, Zeichner A, Kuczmierczyk AR, & Boczkowski J. Famial pain models: the relationship between family history of pain and current pain experience. Pain 1985:21;379-384.

21. Engel GL. "Psychogenic" pain and the pain-prone patient. American Journal of Medicine 1959:26:899-918.

22. Fishbain DA, Goldberg M, Labbe E, Steele R, Rosomoff H. Compensation and non-compensation chronic pain patients compared for DSM-IIII operational diagnoses. Pain 1986:32;197-206.

23. Flor H, Turk DC, & Rudy TE. Relationship of pain impact and significant other reinforcement of pain behaviours: the mediating role of gender, marital status and marital satisfaction. Pain 1989:38;45-50.

24. Frymoyer JW. Magnitude of the problem. In: Weinstein JW & Weisel SW (eds) The Lumbar Spine, Philadelphia: W B Saunders 1990:32-37.

25. Gamsa A, & Vikis-Freibergs V. Psychological events are both risk factors in, and consequences of, chronic pain. Pain 1991:44;271-277.

26. Gamsa A. Is emotional disturbance a precipitator or a consequence of chronic pain? Pain 1990:42;183-195.

27. Gatchel RJ, Mayer TG, Capra P, Diamond P, & Barnett J. Quantification of lumbar function. Part 6: The use of psychological measures in guiding physical functioal restoration. Spine 1986:11;1;36-42.

28. Gil KM, Keefe FJ, Crisson JE, & Van Dalfsen PJ. Social support and pain behaviour. Pain 1987:29;209-217.

29. Grieve EFM. Mechanical dysfunction of the sacroiliac joint. International Journal of Rehabilitation Medicine 1982:5;46-52.

30. Grushka M, Sessle BJ, Milner R. Pain and personality profiles in burning mouth syndrome. Pain 1987:28;155-167.

31. Guest GH & Drummond PD. Effect of compensation on emotional state and disability in chronic back pain. Pain 1992:48;125-130.

32. Gupta M. Is chronic pain a variant of depressive illness? Canadian Journal of Psychiatry 1986:31;241-248.

33. Haldeman S. Presidential Address, North American Spine Society: Failure of the pathology model to predict back pain. Spine 1990:15:;7;718-724.

34. Harkapaa K, Jarvikoski A, Mellin G, Hurri H, Luoma J. Health locus of control beliefs and psychological distress as predictors for treatment outcome in low-back pain patients: results of a 3-month follow-up of a controlled intervention study. Pain 1991:46;35-41.

35. Harper AC, Harper DA, Lambert LJ, et al. Symptoms of impairment, disability and handicap in low back pain: a taxonomy. Pain 1992:50;189-195.

36. Holmes JA, & Stevenson CAZ. Differential effects of avoidant and attentional coping strategies adaptation to chronic and recent onset pain. Health Psychology 1990:9;577-584.

37. Jamison RN, Matt DA & Parris CV. Effects of time-limited vs unlimited compensation on pain behaviour and treatment outcome in low back pain patients. Journal of Psychosomatic Research 1988:32;3;277-283.

38. Javid MJ. Signs and symptoms after chemonucleolysis. A detailed evaluation of 214 worker's compensation and noncompensation patients. Spine 1988:13;12;1428-1437.

39. Jensen MP, Turner JA, Romano JM, & Karoly P. Coping with chronic pain: a critical review of the literature. Pain 1991:47;249-283.

40. Jensen MP, Turner JA & Romano JM. Self-efficacy and outcome expectancies: relationship to chronic pain and coping strategies and adjustment. Pain 1991:44;263-269.

41. Keefe FJ, Crisson J, Urban BJ, & Williams DA. Analyzing chronic low back pain: the relative contribution of pain coping strategies. Pain 1990: 40;293-301.

42. Keefe FJ. Behavioural measurement of pain. In: Chapman CR & Loeser JD (eds.) Issues in Pain Measurement, New York: Raven Press, 1988:405-424.

43. Keefe FJ, Bradley LA, Crisson JE. Behavioural assessment of low back pain: identification of pain behaviour subgroups. Pain 1990:40;153-160.

44. Kelsey JL, White AA, Pastides H, Bisbee GE. The impact of musculoskeletal disorders on the population in the United States. Journal of Bone and Joint Surgery 1979:61A;595-563.

45. Kelsey J, White A, III. Epidemiology and impact on low back pain. Spine 1980;5:2,133-142.

46. Kennedy F. The mind of the injured worker and its effect on disability periods. Compensation Medicine 1946:1;19-24.

47. Krusen EM, Ford DE. Compensation factor in low back injuries. Journal American Medical Association, 1958:166;1128-1133.

48. Kumar S. Cumulative load as a risk factor for back pain. Spine 1990:15; 12;1311-1316.

49. Labbe EE, Fishbain D, Goldberg M, Steele-Rosomoff R, & Rosomoff HL. Compensation and non-compensation pain patients. Responses to the millon behavioural health inventory. Pain Management 1988:(May/June); 133-139.

50. Lawson K, Reesor KA, Keefe FJ, & Turner JA. Dimensions of pain-related cognitive coping: cross-validation of the factor structure of the Coping Strategy Questionnaire. Pain 1990:43;195-204.

51. Leavitt F, Garron DC, McNeill TW, Whisler WW. Organic status, psychological disturbance, and pain report characteristics in low-back-pain patients on compensation, Spine 1982:7;4;398-402.

52. Leavitt F. The physical exertion factor in compensable work injuries. A hidden flaw in previous research. Spine 1992:17;3;307-310.

53. Leavitt F, Garron DC. Validity of a back pain classification scale among patients with low back pain not associated with demonstrable organic disease. Journal of Psychosomatic Research 1976:23;301-306.

54. Lousberg R, Schmidt AJM & Groenman NH. The relationship between spouse solicitousness and pain behaviour: searching for more experimental evidence. Pain 1992:51;75-79.

55. Magni G, Caldieron C, Rigatti-Luchini S, & Merskey H. Chronic musculoskeletal pain and depressive symptoms in the general population. An analysis of the 1st National Health and Nutrition Examination Survey data. Pain 1990:43;299-307.

56. Magni G, Rossi MR, Rigatti-Luchini S, & Merskey H. Chronic abdominal pain and depression. Epidemiologic findings in the United States. Hipanic Health and Nutrition Examination Survey. Pain 1992:49;77-85.

58. Manning MM, & Wright TL. Self-efficacy expectancies, outcome expectancies, and the persistence of pain control in childbirth. Journal of Personality Social Psychology 1983:45;421-431.

59. Mayer TG, Gatchel RJ. & Kishino N. Objective assessment of the spine function following industrial injury: A prospective study with comparison group and one-year follow-up. Spine 1985:10;481-494.

60. McAlary PW, & Aronoff GM. A review of the chronic pain and disability syndrome: Prevalence, contributing factors, detection, prevention and treatment. In: Aronoff GM (ed) Pain Centers: A Revolution in Health Care. New York: Raven Press 1988:201-222.

61. Melzack R, Katz J, & Jeans M. The role of compensation in chronic pain: Analysis using a new method of scoring the McGill Pain Questionnaire. Pain 1985:23;101-112.

62. Mendelson G. Compensation and chronic pain. Pain 1992:48;121-123.

63. Mendelson G. Compensation, pain complaints, and psychological disturbance. Pain 1984:20;169-177.

64. Merskey H. Comments on "Chronic pain as a variant of depressive disease: the pain-prone disorder" Journal of Nervous and Mental Disease 1982:170;409-411.

65. Miller H. Accident Neurosis. British Medical Journal 1961:1;919-925.

66. Morris DB. The Culture of Pain. University of California Press Berkeley, 1991:1.

67. Nachemson A. The lumbar spine: An orthopaedic challenge. Spine 1976:1; 9-21.

68. Nagy VT, Wolfe GR. Cognitive predictors of compliance in chronic disease patients. Medical Care 1984:22;912-921.

69. Naliboff BD, Cohen MJ, Swanson GA, Bonebakker AD, & McArthur DL. Comprehensive assessment of chronic low back pain patients and controls: Physical abilities, level of activity, psychological adjustment and pain perception. Pain 1985:23;121-134.

70. Naliboff BD, Cohen MJ, & Yellin A. Does the MMPI differentiate chronic illness from chronic pain? Pain 1982:13;333-341.

71. National Centre for Health Statistics: Physiotherapy office visits: National Ambulatory Medical Care Survey: United States 1980-81. Advance Data from Vital and Health Statistics, no.120, D.H.S.S. publication # (PHS) 86-1250. Public Health Service, Hyattsville, MD, July, 11, 1986.

72. Osterweis M, Kleinman A, Mechanic D. Pain and Disability. Clinical, behavioral, and public policy perspectives. Washington: National Academy Press, 1987.

73. Parker N. Accident Neurosis. Medical Journal of Australia 1970:2;362-365.

74. Pilowsky I. Abnormal illness behaviour (Dysnosognosia). Psychotherapy and Psychosomatics 1986:46;76-84.

75. Pither CE, Nicholas MK.The identification of iatrogenic factors in the development of chronic pain syndromes: abnormal treatment behaviour? In: Bond MR, Charlton JE, & Woolf CJ (eds). Proceedings of the VIth World Congress on Pain Elsevier, Netherlands: Elsevier, 1991:429-434.

76. Polatin PB, Gatchel RJ, Barnes D, Mayer H, Arens C, & Mayer TG. A psychosociomedical prediction model of response to treatment by chronically disabled workers with low-back pain Spine 1989:14;9;956-961.

77. Polatin PB, Kinney RK, Gatchel RJ, Lillo E, Mayer TG. Psychiatric illness and chronic low-back pain. Spine 1993:18;1,66-71.

78. Quebec Task Force on Spinal Disorders Report. Spine 1987:12;S7;8-59.

79. Reesor KA, Craig KD. Medically incongruent chronic back pain: physical limitations, suffering, and ineffective coping. Pain 1988:32;35-45.

80. Rollman GB. Measurement of experimental pain in chronic pain patients: Methodological and individual factors. In: Mezack R (ed) Pain Measurement and Assessment. New York: Raven Press 1983:251-258.

81. Romano JA, Turner JA. Chronic pain and depression: does the evidence support a relationship. Psychological Bulletin 1985:97;18-34.

82. Rosensteil AK, Keefe FJ. The use of coping strategies in chronic low back pain patients: relationship to patient characteristics and current adjustment. Pain 1983:17;133-144.

83. Rossi U and Pernak J. Low back pain: the facet syndrome. In: Lipton S. et al (eds). Advances in Pain Research and Therapy 73; York: Raven Press, 1990:231-244.

84. Rowat KM, Knapfl KA. Living with chronic pain: the spouse's perspective. Pain 1985:23;259-271.

85. Roy R. Engel's pain-prone disorder patient: 25 years after. Psychotherapy and Psychosomatics 1985:43;126-135.

86. Rudy T, Kerns R, Turk D. Chronic pain and depression: toward a cognitive-behavioural mediation model. Pain 1988:35;129-140.

87. Rudy TE, & Turk DC. Chronic pain and depression. II. Five fashionable fads. Pain Management 1988:(Jan/Feb);7-17.

88. Schnurr RF, Brook RI, Rollman GB. Psychosocial correlates of temporomandibular joint pain and dysfunction. Pain 1990:42;153- 165.

89. Schwartz L, Slater MA, Birchler GR, Atkinson JH. Depression in the spouses of chronic pain patients: the role of patient pain and anger, marital satisfaction. Pain 1991:44;61-67.

90. Sherman RA, Sherman CJ, Bruno GM. Psychological factors influencing chronic phantomb limb pain: an analysis of the literature Pain 1987:28;285-295.

91. Simmonds MJ, & Kumar S. The bases of low back pain. Neuro-orthopaedics 1992:13;1-14.

92. Snook SH, Jensen RC. Cost. In: Pope MH, Frymoyer JW, & Andersson G (eds.) Occupational Low Back Pain. New York: Praeger Press, 1984.

93. Spengler DM, Bigos SJ, Martin NA, Zeh J, Fisher L, Nachemson A. Back injuries in industry: A retrospective study. I. Overview and cost analysis. Spine 1986:11;241-245.

94. Steig RL. The cost-effectiveness of pain treatment: who cares? The Clinical Journal of Pain. 1990:6;301-304.

95. Sternbach RA & Timmermans G. Personality changes associated with reduction of pain. Pain 1975:1;177-181.

96. Sternbach RA. Pain Patients: Traits and Treatments New York: Academic Press, 1974.

97. Sternbach RA. Survey of pain in the United States: the Nuprin pain report. Clinical Journal of Pain 1986:2;49-53.

98. Tait RC, Chibnall JT, & Richardson WD. Litigation and employment status: effects on patients with chronic pain. Pain 1990:4;37-46.

99. Tate DG. Workers disability and return to work. American Journal of Physical Medicine and Rehabilitation 1992:71;2;92-96.

100. Tauschke E, Merskey H, Helmes E. Psychological defence mechanisms in patients with pain. Pain 1990:40;161-170.

101. Tearnan BH, Cleeland CS. The attitudes of physicians toward chronic pain patients. Pain Management 1988:180-184.

102. Tearnan BH, Cleeland CC. The attitudes of physicians toward chronic pain patients. Pain Management 1988:(July/Aug);180-184.

103. Turk DC, Flor H, & Rudy TE. Pain and families. I. Etiology, maintenance, and psychological impact. Pain 1987:30;3-27.

104. Turk DC, Rudy TE, & Steig RL. Chronic pain and depression. I. "Facts". Pain Management 1987:(Nov/Dec);17-25.

105. Turk DC, Salovey P. "Chronic pain as a variant of a depressive disease" a critical reappraisal. Journal of Nervous and Mental Disease 1984:172;398-404.

106. Turner JA, Clancy S. Strategies for coping with chronic low back pain: relationships to pain and disability. Pain 1986:24:355-364.

107. Vollin E, Lai D, McKinney S, Loeser JD. When back pain becomes disabling: a regional analysis. Pain 1988:33;33-39.

108. Waddell G, Main CJ, Morris EW, Di Paola M, Gray ICM. Chronic low-back pain, psychologic distress, and illness behaviour. Spine 1984:9;2,209-213.

109. Waddell G, Newton M, Henderson I, Somerville D, Main CJ. A fear-avoidance beliefs questionnaire (FABQ) and the role of fear-avoidance beliefs in low back pain and disability. Pain 1993:52;157-168.

110. Waddell G, Pilowsky I, & Bond MR. Clinical assessment and interpretation of abnormal Illness behaviour in low back pain. Pain 1989: 39;41-53.

111. Waddell G, Bircher M, Finlayson D, Main C. Symptoms and signs: physical disease or illness behaviour? British Medical Journal 1984:289:739-740.

112. Wade JB, Dougherty LM, Hart RP, & Cook DB. Patterns of normal personality structure among chronic pain patients. Pain 1992:48;37-43.

113. Wade JB, Dougherty LM, Hart RP, Rafii A, Price DD. A canonical correlation analysis of influence of neuroticism and extraversion on chronic pain, suffering, and pain behaviour. Pain 1992:51;67-73.

114. Wallston KA, Wallston BS, & De Vellis R. Development of the multidimensional health locus of control (MHLC) scales, Health Education Monogram 1987:6;160-170.

115. Watson D. Neurotic tendencies among chronic pain patients: an MMPI item analysis. Pain 1982:14;365-385.

116. Weighill VE, Buglass D. An updated review of compensation neurosis. Pain Management 1989:(Mar/Apr);100-105.

117. Weighill VE, "Compensation Neurosis": A review of the literature. Journal of Psychosomatic Research 1983:27;97-104.

118. Widerstrom EG, Aslund PG, Gustafsson LE, Mannheimer C, Carlsson SG, & Andersson SA. Relations between experimentally induced tooth pain threshold changes, psychometrics and clinical pain relief following TENS. A retrospective study in patients with longlasting pain. Pain 1992:1;281-287.

119. Ziporyn TL. Disability evaluation: A fledgling science? Journal of the American Medical Association 1983:250;873-880.

120. Åstrand NE. Medical, psychological, and social factors associated with back abnormalities and self reported back pain: a cross sectional study of male employees in a Swedish pulp and paper industry. British Journal of Industrial Medicine 1987:44;327-336.

CHAPTER FOUR

INFLUENCE OF KNOWLEDGE IN THE ASSESSMENT OF LOW BACK PAIN[3]

Introduction

Low back pain (LBP) has been a human affliction since ancient times (19). But the high rate of disability due to LBP, is a relatively recent phenomenon. In industrialised countries disability due to LBP has increased at a rate 14 times greater than the population growth (10). The influence of compensation on LBP and disability is one of the most controversial issues in the treatment of LBP (30).

LBP causes disruption of work and social activity and leads to tremendous utilization of healthcare services. Many occupational groups make an excellent living by treating LBP (27). Medical costs such as physicians' fees and drugs are much higher in back injuries compared to other musculoskeletal conditions (12). They will also be higher in compensation compared to non-compensation patients if the recovery period is longer and medical treatments are continued. Krusen and Ford (14) reported that compensation patients received significantly more treatments than those not on compensation (14). Recent changes in authorization procedures by the Alberta WCB for physical therapy have been designed to provide checks on potential overtreatment (personal communication Alberta WCB). However, the potential for overtreatment is found within all health professions. Peters et al (19) revealed that 73% of patients attending a pain clinic had seen a physical therapist, 46% an orthopaedic surgeon, 51% an anesthetist, 50% a rheumatologist, 36% a neurologist, and 21% a psychiatrist (21). This is important information because LBP is the most common reason that patients attend pain clinics (27) and so it is reasonable to assume that these number of referrals would apply to many of the patients with chronic LBP. These figures help to explain why the total cost of LBP in the United States is estimated at $85 billion (6). This is not to suggest that clinicians overtreat for personal financial reward. However, it has been noted that physicians tend to overestimate their own effectiveness and underestimate the risks of treatment (17). The risk of unnecessary treatment is iatrogenically induced disability (13,8) and dependence on the health care system.

The traditional medical model has tended to focus on physical factors and this has contributed to its failure to halt the disability epidemic (11). The multiplicity of empirical treatments and the persistence and recurrance of the problem are further testimony to this failure (26). In a study addressing the appropriateness of medical treatments, only 3 out of 89 patients had not had

---

[3]A version of this chapter has been submitted for publication. Simmonds, Kumar and Lechelt 1993. Archives of Physical Medicine.

some form of inappropriate investigation or treatment (22). No treatment is better than inadequate treatment (14). Unfortunately, at the present time many treatments are not based on empirical evidence and are applied to non-specific or undiagnosed problems of the back. So the question of which treatment is appropriate for what condition, when, and for how long, is open to interpretation. But, it should be remembered that most cases of benign LBP settle with no treatment (24).

There is a growing recognition of the importance of psychosocial factors in determining not only the effects of LBP, (7,4,16), but also the response to treatment (26).

It is acknowledged that all treatments have a placebo or non-specific effect. The extent of which is variable. Factors such as the quality of the relationship and the expectancies of clinician and client, will influence treatment outcome through non-specific means. In fact the art of medicine and physiotherapy is based on non-specific effects (20). Basmajian (3) has stated that the main virtue of rehabilitation is the intensive relationship formed between professionals and their patients. Given that the placebo effect is enhanced by the degree of understanding and enthusiasm of the clinician and their beliefs in the treatment efficacy (9). It makes intuitive sense, that the quality of the relationship will influence the non-specific treatment effects and thus the rehabilitation outcome.

Placebo effects of physiotherapy have been demonstrated in numerous studies (for review see 25). However, the placebo effect is not well understood theoretically (28). Both conditioning and expectancies contribute to the placebo effect (29). Therefore past experiences and current beliefs and biases will influence the expectancies and thus the efficacy of treatment.

This point may be relevant to the controversy that surrounds the negative effect of compensation, on treatment outcome. The presence of negative bias towards compensation patients has been reported. Melzack (18) stated that the "compensation recipient with LBP is the pariah of modern medicine". Whilst Leavitt (15), has noted that the literature which casts a negative image on the industrial worker is far less conclusive than might be inferred from the biases of this group embedded in print. Given that a negative bias is present, it is reasonable to suggest that it influences the enthusiasm and expectancy of treatment efficacy and in this manner the treatment outcome is somewhat predetermined. This is the nature of a self-fulfilling prosphesy.

Whether this actually occurs has not been tested directly. It is not clear whether the expectancy of outcome is influenced by compensation status. If this is so, then it has important implications for all clinicians who treat compensation claimants. The aim of the present study was to address this issue and to determine whether prior knowledge of compensation status influences: a. physical assessment findings and b. prognosis of physical therapy outcome.

METHODOLOGY

Overview

This study was a controlled randomised double blind study. Sixty-nine physical therapists (PTs) blind to the research question observed three pre-recorded videotapes of patients undergoing a clinical back assessment. The PTs recorded their assessment findings during the observation period and in addition made judgements about the subjects prognosis.

Physical Therapist Observers

Sixty nine PTs participated as observers in this experiment. Their primary employment was in acute care hospitals (n=20), rehabilitation centres (n=21) and private physiotherapy clinics (n=21). Seven subjects were graduate students or faculty members in a physical therapy programme at the University of Alberta. The mean age of the PTs was 33.8 years with a range of 21 and 59 years. The mean time for which they had practiced physical therapy was 10 years, with a range from less than 1 year to a maximum of 29 years.

Videotaped Subjects

A total of seven subjects were videotaped. One male subject was assymptomatic and his tape was used for training purposes. Two subjects, one male and one female had complaints of very mild backache and no other symptoms. Two subjects, one male and one female had complaints of moderate to severe pain in their back and leg. Two subjects both male, were actors who were coached to fake back and leg pain (see Table 4.1). All subjects completed a Roland disability questionnaire (23). They were advised about the purpose of the study and signed an informed consent prior to participation.

Information Packages

The independant variable was information (INFO) of which there were three levels; WCB, NWCB and CON for each of the six subjects. The CON INFO acted as control and no information was provided about the subjects medical or social history nor about the subjective complaints of back pain. The WCB and NWCB INFO packages were similar in their inclusion of medical history and subjective complaints of pain for each subject and the Roland questionnaire which had been completed by the subject. The WCB INFO package included a statement that the subject viewed on videotape was in receipt of workers compensation. The NWCB INFO package included a statement that the subject was not in receipt of workers compensation. Apart from the issue of compensation status, the history of back pain included in the INFO package was the true history for each of the four patients. The actors were assigned a typical history of LBP.

Table 4.1. Characteristics of the videotaped subjects

Subject	Gender	Occupation	Age	Severity of symptoms
1	Female	Teacher	51	Mild back pain No leg pain
2	Female	Nurse	25	Moderate back and leg pain
3	Male	Maintenance man	35	Moderate back and leg pain
4	Male	Maintenance man	39	Minimal occasional back pain
5	Male	Actor and construction worker	24	Moderate back and leg pain
6	Male	Actor and student	24	Moderate back and leg pain

Videotape procedure

A standardised lumbar spine assessment of all subjects was performed by an experienced physical therapist. The procedure was as follows. Subjects were first observed in a seated position, they then stood up and walked approximately 70 feet towards the camera, before turning around and returning to their seat. The subjects were next seen standing and observation was allowed from anterior, posterior, right and left sides. Next, spinal range of motion in each plane was carried out, and subjects were observed from both global and close up perspectives. In order to facilitate observation, the spinous process from L1 to S1, and the posterior superior iliac spines, were identified with colored dots. A straight leg raise (SLR) and prone knee flexion test was then carried out on each leg. Finally, testing of dermatomes and myotomes was carried out. These were not scored in any way. They were simply done because it is a standard component of a lumbar spine assessment and the test provided more observation time for the PT observer.

Experimental procedure

The final edited version of each subjects videotape was between 10 and 13 minutes. Ten copies of each tape were made so that several sets of tapes could be circulated simultaneously. Each PT viewed three tapes. The randomization procedure for subjects and INFO was carried out in the following manner. First, a random assignment of subjects was carried out and arranged into sets of three. Next, assignment of INFO (WCB, NWCB or CON) was carried out for each subject. Thus each PT would observe three different subjects. The INFO assignment may have been the same for all three subjects, or there may have been a combination of INFO assignments.

The INFO for each subject was placed into a sealed envelope along with the data collection forms. The videotape ID number was placed on the outside of the envelope in order that the corresponding videotape could be provided to the PT observer. The INFO condition was not identified on the outside of the envelope to maintain blindness. Each PT was provided with the training tape and written instructions, three subject tapes with the corresponding INFO package and the data collection forms.

Observation Procedure

The PTs viewed the tapes alone. They were given verbal and written instructions about viewing and scoring the videotapes. They were allowed to watch the training tape more than once, but the subject tapes were only viewed once. There was no specified order in which to view the three tapes. Observers were advised to pause the tape in order to score an item, but not to review a section more than once. This procedure ensured that all observers watched and scored the tape in a standardised manner.

Data was collected on a number of items. Lumbar range of motion (ROM) was judged as a percentage of normal for each motion (normal was determined as 100%) whereas SLR was measured in degrees. Pilot testing revealed good reliability between judges ($r > .9$) for ROM measures. Gait, posture, impairment (IMPAIR), disability (DISAB), short and long term prognoses (SHTPROG and LTPROG respectively) were measured using a 0 - 10 numerical rating scale. The "0" end point was marked as "normal" for gait and posture; "no impairment" for the IMPAIR scale; and "no disability" for DISAB, SHTPROG (1 month) and LTPROG (1 year) scales. The "10" end point was marked as "totally abnormal" for gait and posture, and "total impairment" or "total disability" for the other measures. Finally, PTs were asked whether the subject needed physical therapy or whether they would refer them to a specialist. These were anwered either "yes" or "no". A following question asked how likely it was that the patient would benefit from PT (PTBEN). The latter question was answered using an 11 point numerical rating scale with "0" marked as "unlikely to benefit" and "10" marked as "very likely to benefit."

Data Analysis

The effect of INFO was analysed using an overall 6 x 3 univariate ANOVA (six levels of subjects and 3 groups) and a series of one way ANOVAs across INFO groups. The relationships between dependent variables were analysed using Pearson correlation coefficients. The level of significance was set at $p < .05$ for all analyses.

RESULTS

The effect of INFO

Descriptive statistics were computed for dependent variables for each subject in each INFO group. These data are presented in Tables 4.2, 4.3 and 4.4.

The effect of INFO was assessed on each subject across groups using 1-way ANOVAs. Although the number of comparisons suggest that some differences may occur by chance, certain trends are present. Similar tendencies were evident across the LBP subjects but not the actors. Subjects 1 and 4 both exhibited mild signs of back problems. For subject 1, a female, The ANOVA revealed a significant effect of INFO for PTBEN ($DF_{2,30}$ $F = 4.56$, $p < .02$), SHTPROG ($DF_{2,29}$ $F = 3.53$, $p < .05$), IMPAIR ($DF_{2,30}$ $F = 3.39$, $p < .05$) and DISAB ($DF_{2,30}$ $F = 4.52$, $p < .02$). Post-hoc Tukey tests revealed that the control group was significantly different from the other two groups (see Table 4.4). Subject 4 was a male and the ANOVA revealed a significant effect for SHTPROG ($DF_{2,32}$ $F = 14.82$, $p < .00001$), IMPAIR ($DF_{2,33}$ $F = 4.10$, $p < .05$), DISAB ($DF_{2,31}$ $F = 6.09$, $p < .005$). Post-hoc tests revealed that the NWCB group was significantly different from the other two groups (see Table 4.4).

Subjects 2 and 3 both exhibited signs of marked dysfunction due to LBP. Subject 2 was a female and the ANOVA revealed a significant of INFO on right SLR ($DF_{2,30}$ F = 3.63, p <.05) in the WCB group. Subject 3 was a male and the ANOVA revealed significance for PTBEN ($DF_{2,32}$ F = 9.51, p <.001), SHTPROG ($DF_{2,32}$ F = 10.84, p <.0005), LTPROG ($DF_{2,32}$ F = 6.41, p <.005) in the NWCB group.

Subjects 5 and 6 were both male actors. Subject 5 demonstrated marked back dysfunction. The ANOVA revealed a significant effect of INFO on gait ($DF_{2,36}$ F = 3.56, p <.05), and SHTPROG ($DF_{2,38}$ F = 3.63, p <.05) in the WCB group. Subject 6 demonstrated milder back dysfunction and the ANOVA was significant for flexion, ($DF_{2,25}$ F = 5.56, p <.01), in the WCB group. Right rotation ($DF_{2,25}$ F = 4.75, p <.02), and LSLR ($DF_{2,24}$ F = 5.36, p < .01) were significantly different in the NWCB group.

As noted above, subjects 1 and 4 both demonstrated mild back symptoms. The data from these subjects was pooled and the effect of INFO across this subgroup was analysed with a one - way ANOVAs. Significant differences were revealed for PTBEN ($F_{2,66}$ = 5.5, p <.01), IMPAIR ($F_{2,66}$ = 7.1, p <.001) and DISAB ($F_{2,64}$ = 10.0, p <.0005) in the CON group. For SHTPROG ($F_{2,64}$ = 12.5, p <.0001), all groups were significantly different from each other. The LTPROG ($F_{2,65}$ = 3.7, p <.05) was different in the WCB compared to the CON group. Subjects 2 and 3 were also similar in that both had complaints of marked pain. These subjects were also analysed as a subgroup. The ANOVA revealed significant differences for PTBEN ($F_{2,66}$ = 8.5, p <.0005) and LTPROG ($F_{2,65}$ = 4.3, p <.01) in the NWCB group. For SHTPROG ($F_{2,66}$ = 4.6, p <.01) the WCB group was different from the NWCB group.

Correlations between variables

Pearson correlation coefficients were computed for the group as a whole as well as across INFO groups. The ROM correlations within each group reflected a similar pattern to that of the group as a whole (n = 164). All ROM were significantly correlated at the p <.001 level. The number of cases analysed in the WCB, NWCB and CON groups were 56, 55 and 53 respectively. The fact that individual therapists could have contributed more than once to a group may have inflated the correlations. The lowest ROM correlations were between rotatation and extension (r = 0.73 in the NWCB group to r = 0.80 in the WCB group. The highest correlations occurred between rotation and sideflexion to the same side. The range was r = 0.85 to r = 0.94, p <.001).

In general the relationships between ROM and IMPAIR, DISAB, LTPROG and SHTPROG were negative. The strongest associations were between IMPAIR and all ROM measures in the control group (r = -0.78 to -0.93, p <.001). The weakest were in the WCB group (range r = -0.65 to -0.69, p <.001).

There was little association between LTPROG and ROM in the total sample, but the level of association differed between groups. It was highest in the CON group ($r = -0.33$ to -0.46, $p < .01$ to $p < .001$) and lowest in the NWCB group ($r = 0.07$ to -0.18). Similar group differences were found in the relationship between PTBEN and ROM. The correlation coefficients were close to zero in the WCB group but ranged between $r = -0.36$ and -0.42, ($p < .01$) in the CON group.

Gait was significantly related to DISAB but there were differences in the strength of the relationship across groups, $r = .89$, .71 and .75, in the CON, WCB and NWCB groups respectively. The relationship between DISAB and posture was weaker but showed a similar pattern, $r = .70$, .53 and .55, in the CON, WCB, and NWCB groups respectively.

The relationships between DISAB and IMPAIR were strong and ranged from $r = 0.79$ in the WCB group to $r = 0.93$ in the CON group ($p < .001$). However, there was a difference across groups in the relationship between DISAB and LTPROG. In the CON group the r value was 0.52 which was significant at the $p < .001$ level. In the WCB group the r value was 0.40, significant at the $p < .01$ level, whilst in the NWCB group it was 0.16 and was non significant. The IMPAIR/LTPROG relationship also varied across groups. It was highest in the CON group ($r = 0.41$ ($p < .01$), lowest in the WCB group ($r = 0.04$) and was $r = 0.20$ (n.s) in the NWCB group. SHTPROG was generally related to IMPAIR and DISAB especially in the CON group, $r = 0.83$ ($p < .001$). There was a reasonable correlation between the Roland disability scale and that judged by the PTs $r = .70$ ($p < .001$).

Finally, PTs were asked whether the subject needed physical therapy. In the WCB group 92% or 66 PTs said yes, this compared to 79% or 56 PTs in the NWCB group and 63% or 40 PTs in the CON group. PTs were also asked whether they would refer the subject to another professional. In the WCB group 53% or 38 PTs said yes, compared to 48% or 34 PTs in the NWCB group and 42% or 27 PTs in the CON group. The majority of referrals were to orthopaedic surgeons or psychologists.

Table 4.2. Group means (percentage of normal) and standard deviations of range of motion for each subject.

Subject	Variable	WCB	NWCB	CON
1	FLEXION	98.0(6.3)	76.0(21.9)	85.4(26.6)
	EXTN	87.0(20.6)	89.7(11.6)	77.7(29.8)
	LSDFLEX	88.0(12.1)	90.0(12.4)	71.6(28.6)
	LROTN	93.0(10.6)	83.7(19.0)	85.0(16.1)
	RSDFLEX	83.0(13.8)	94.6(6.5)	77.3(26.4)
	RROTN	89.0(11.2)	89.6(10.7)	87.7(12.9)
2	FLEXION	38.6(8.4)	29.2(11.0)	36.4(19.0)
	EXTN	26.4(11.8)	38.7(20.0)	43.2(29.4)
	LSDFLEX	47.9(18.3)	49.2(18.4)	49.5(14.7)
	LROTN	50.9(16.4)	55.8(20.2)	56.4(19.0)
	RSDFLEX	42.0(10.6)	47.5(11.6)	40.9(15.5)
	RROTN	44.1(19.8)	45.8(13.6)	45.4(14.9)
3	FLEXION	9.1(9.9)	10.4(9.2)	14.1(10.7)
	EXTN	0.9(3.0)	2.7(3.9)	3.2(4.6)
	LSDFLEX	11.4(12.3)	6.2(4.0)	13.6(21.1)
	LROTN	3.6(8.9)	4.6(6.3)	7.3(11.5)
	RSDFLEX	4.5(4.7)	2.3(3.3)	0.9(2.0)
	RROTN	8.2(20.8)	1.3(3.0)	1.8(3.4)
4	FLEXION	83.2(29.2)	84.1(18.7)	88.6(14.3)
	EXTN	61.8(29.4)	59.5(33.8)	49.1(29.0)
	LSDFLEX	77.3(20.1)	87.7(11.9)	85.0(14.6)
	LROTN	82.1(17.7)	83.2(14.2)	84.1(15.3)
	RSDFLEX	84.3(18.1)	93.6(8.1)	92.3(14.7)
	RROTN	85.3(18.4)	80.9(17.1)	90.0(14.3)
5	FLEXION	12.3(5.9)	12.0(7.9)	15.4(5.2)
	EXTN	7.3(7.7)	11.7(22.8)	4.1(4.4)
	LSDFLEX	15.3(9.1)	17.6(10.0)	15.9(6.2)
	LROTN	32.6(16.9)	36.1(17.4)	32.3(10.3)
	RSDFLEX	9.2(6.6)	7.0(5.6)	5.4(4.1)
	RROTN	23.0(11.1)	29.0(16.1)	25.9(9.2)
6	FLEXION	59.5(13.3)**	38.7(15.2)	38.8(19.5)
	EXTN	40.2(25.4)	21.9(16.9)	31.7(22.1)
	LSDFLEX	56.8(14.7)	36.9(13.6)	53.9(26.6)
	LROTN	53.2(19.1)	32.5(21.5)	36.7(19.7)
	RSDFLEX	39.1(12.0)	23.1(15.7)	33.3(23.3)
	RROTN	37.5(16.9)	16.9(10.3)**	37.8(18.9)

Abbreviations: EXTN = extension, (R)LSDFLEX = Side flexion to the left or (right) side, (R)LROTN = rotation to the left or (right) side;
** 1 way Anova significant $p < .01$

Table 4.3. Group means and standard deviations of gait, posture and SLR for each subject.

Subject	Variable	WCB	NWCB	CON
1	GAIT	2.7(3.1)	2.2(2.3)	1.3(1.4)
	POSTURE	3.2(1.6)	3.7(2.1)	2.6(1.4)
	RSLR	90.0(2.3)	92.0(4.5)	91.1(6.0)
	LSLR	87.8(2.5)	92.0(4.5)	90.5(6.3)
2	GAIT	6.4(1.8)	5.1(2.2)	5.2(1.9)
	POSTURE	5.3(2.0)	3.2(2.3)	3.9(2.5)
	RSLR	60.9(9.4)*n	50.8(6.3)	52.5(12.3)
	LSLR	69.5(8.5)	65.0(8.1)	63.0(9.8)
3	GAIT	8.3(1.3)	8.3(1.0)	8.0(1.4)
	POSTURE	8.5(1.2)	8.5(1.0)	7.8(1.5)
	RSLR	10.0(9.7)	20.8(9.5)	15.5(11.4)
	LSLR	30.5(10.7)	30.8(10.8)	36.0(10.2)
4	GAIT	0.5(0.7)	1.1(0.8)	0.7(0.8)
	POSTURE	3.1(2.6)	2.8(1.1)	2.4(1.4)
	RSLR	82.8(3.2)	84.1(3.7)	86.5(5.8)
	LSLR	83.2(3.7)	83.6(3.9)	85.0(6.6)
5	GAIT	8.7(0.8)*c	8.2(1.2)	7.3(1.9)
	POSTURE	5.6(3.1)	4.7(2.5)	5.8(2.5)
	RSLR	40.3(11.6)	41.3(7.4)	40.0(7.7)
	LSLR	54.3(7.8)	56.3(11.6)	52.7(12.3)
6	GAIT	5.1(1.9)	5.0(2.7)	5.7(1.4)
	POSTURE	6.0(1.5)	5.3(2.1)	6.0(0.5)
	RSLR	48.6(6.7)	45.0(3.8)	47.5(3.8)
	LSLR	80.4(5.2)	75.0(5.3)**	83.1(4.5)

Abbreviations: (R)LSLR = (right) or left straight leg raise
Legend: Gait and posture measured on a 0-10 numerical rating scale, 0 = normal, 10 = totally abnormal
SLR measured in degrees
Statistical test 1 way Anovas
** = significant $p < .01$
*c = significantly different from the CON group
*n = significantly different from the NWCB group

Table 4.4. Group means and standard deviations of impairment, disability and prognosis for each subject.

Subject	Variable	WCB	NWCB	CON
1	PTBEN	6.3(3.2)	5.9(2.2)	3.0(2.9)**
	IMPAIR	2.2(2.0)	2.4(1.6)	0.8(1.1)*n
	DISAB	2.2(1.8)	2.6(2.0)	0.6(0.8)*
	SHTPROG	3.7(2.6)	3.3(2.7)	1.1(1.5)*
	LTPROG	3.3(3.6)	2.9(2.3)	0.9(1.7)
2	PTBEN	7.6(2.8)	8.6(1.2)	7.3(1.8)
	IMPAIR	5.4(2.5)	4.7(1.6)	5.3(1.9)
	DISAB	5.6(2.2)	5.5(1.7)	4.9(1.9)
	SHTPROG	4.1(2.4)	3.4(1.8)	4.5(2.5)
	LTPROG	1.0(1.3)	0.6(0.9)	2.3(2.6)
3	PTBEN	5.0(2.8)	8.8(1.8)***	5.1(2.7)
	IMPAIR	8.5(2.1)	8.5(1.0)	8.1(1.0)
	DISAB	9.0(0.9)	8.3(0.8)	7.9(1.7)
	SHTPROG	8.8(1.2)	4.8(2.4)***	6.8(2.3)
	LTPROG	5.5(2.7)	2.1(1.4)*w	3.8(2.8)
4	PTBEN	6.6(3.0)	6.4(2.9)	4.4(4.2)
	IMPAIR	2.3(2.0)**c	1.7(1.3)	0.6(0.6)
	DISAB	3.2(2.4)**c	2.1(2.5)	0.4(0.5)
	SHTPROG	4.1(2.3)**	1.5(1.5)	0.5(0.7)
	LTPROG	1.8(1.6)*c	0.9(2.0)	0.4(1.2)
5	PTBEN	5.5(2.4)	5.3(3.1)	5.5(3.1)
	IMPAIR	6.7(2.3)	7.2(1.6)	7.7(1.1)
	DISAB	7.8(1.6)	7.2(1.9)	7.8(1.2)
	SHTPROG	7.2(1.8)	6.5(1.9)	5.3(1.6)*w
	LTPROG	3.6(2.9)	3.6(2.5)	3.2(2.5)
6	PTBEN	7.8(2.3)	8.5(1.7)	8.2(3.0)
	IMPAIR	5.7(1.9)	5.1(1.4)	4.9(2.0)
	DISAB	5.6(1.6)	5.2(1.2)	5.7(1.8)
	SHTPROG	4.4(2.4)	4.5(1.8)	3.4(1.3)
	LTPROG	1.2(0.9)	1.4(1.2)	2.4(3.9)

Abbreviations: PTBEN = expected benefit from physical therapy 0 = no benefit, 10 = very likely to benefit; IMPAIR = impairment 0 = no impairment, 10 = total impairment; DISAB = disability 0 = no disability, 10 = total disability; SHTPROG = prognosis of disability in 1 month, 0 = no disability, 10 = total disability; LTPROG = prognosis of disability in 1 year, 0 = no disability, 10 = total disability

Statistical tests - 1 way Anovas

*** $p < .001$ *c = significantly different from control group

** $p < .05$, *w = significantly different from WCB group

*n = significantly different from non-WCB group

DISCUSSION

There may be some limitations in this study based on the use of videotape methodology. No verbal communication nor palpation was allowed between the therapist and the patient. Whilst a different methodology may have been more realistic clinically, it could have confounded the results due to variability in communication and palpation skills. Videotape methodology ensured control of potentially confounding information. This strengthens the assertion that prior knowledge influenced the differential judgements in the prognostic variables. Overall there was little effect of prior information on physical assessment findings such as ROM or SLR. However, there was evidence of this information influencing more complex judgements such as impairment, disability and prognosis.

In terms of assessment findings, no obvious trend emerged which was robust across all subjects and all INFO groups. This is a reassuring finding and suggests that little bias enters into the physical assessment of patients with LBP. These measures are relatively simple and are based primarily on observation. They require little interpretation or analysis.

There was an effect of information on judgements of disability and prognosis. Scoring of these measures is more complex and requires some interpretation of information. In this study, the information utilised in order to make a judgement could have been acquired in any of three ways.

1. Information acquired by observation of the subject.
2. Information acquired by reading the medical history.
3. Information acquired by prior experience.

The first two information sources were controlled while the latter was not.

Prior experience was not controlled and neither was it assessed in depth. The age and years of professional experience was measured, and there was no difference between groups on these measures. However, it is acknowledged that neither age nor years of experience is an adequate measure of prior experience. Specific experiences which could confound the results of this study, pertain to the PTs previous experience with WCB clients. If particularly outstanding positive or negative interactions had occurred, this could have biased their view. The fact that there were essentially no differences between INFO groups, suggests that this was not the case.

Physical assessment

Prior experience should have minimal effect on ROM measures, given that ROM can be measured using observational skills only. Moreover, clinical experience does not generally lead to more accuracy (5). Minimal interpretation of the observed information is needed to determine the ROM. It can be seen from Table 4.2 and 4.3 on the ROM, gait and posture variables, that there are

few significant differences either within subjects or across groups. These measures seem to be influenced little by the knowledge of medical history, lending support to the argument that ROM judgements are based only on observation. Moreover, the fact that the CON group which had no information, did not differ from the other two groups, supports this contention. Finally, it appears that the knowledge provided in the INFO package to the WCB and NWCB groups was either not used or it had minimal influence on the decision.

Although some differences do exist they are present only for one of the actors. The group differences in this case may be explained by inconsistencies or apparent exaggeration in his actions. The PTs may have observed these inconsistencies and exaggerations and perhaps their suspicions were confirmed if he was deemed to be a WCB claimant. If this is the case then it appears that confirmation of beliefs is sought but only when some element of suspician occurs, or when there is inconsistent or inadequate information. This contention receives more support in regards to judgements of impairment.

Impairment and disability

There was an effect of INFO on judgements of impairment and disability. This can best be explained in terms of individual subjects. In regards to judgements of impairment, the INFO only influenced judgements for those subjects who exhibited very mild signs of LBP. This suggests that if there are very obvious visual signs of LBP, then other information is either not utilised or not given as much importance, in making a judgement of impairment. If however, the signs of LBP are more subtle, then more information is sought and utilised in decision making. This seems to be a reasonable assertion given that it is the CON group which differs from the other two INFO groups. The positive finding here is that although the WCB group scored higher in terms of impairment, there is no difference between WCB and NWCB groups indicating minimal effects of this bias.

Impairment represents a higher level of judgement than ROM. The definition of impairment is that it is based on physical restriction only. This definition was provided to the PTs on the data collection sheet. A judgement of impairment requires the collection and synthesis of several observations in order to arrive at a decision. The measurements normally used in determining impairment are ROM measures (1). It appears from the data that PTs are using these measures in order to formulate their judgement of impairment. Further confirmation of this, stems from the high correlations between ROM measures and impairment (-.78 to -.93) especially in the CON group.

Theoretically, disability is defined in terms of vocational and social limitations which stem from impairment, this information was provided to the PTs on the data collection forms. In practical terms, disability and impairment are frequently used interchangably. In this study, judgements of disability did not differ from judgements of impairment. The grand mean for impairment was 29.2 and for disability it was 29.8. The correlation between impairment and

disability was between .79 and .93 within the three groups. This may reflect the fact that impairment and disability are thought of in similar terms.

Prognosis of disability

A different picture emerges from the judgements made on disability and prognosis. Here the WCB group tends to fare worse and this is true for all subjects. Disability determination is a measure of the level of function. In order to judge the level of function, the PTs probably utilised the information that was provided to them, about the subject. It is at this level that expectancies appear to play a greater role in influencing judgements. These results are in agreement with those of Ashton et al (2) who found that subjective measurements which were more open to interpretation were more prone to the biasing effect of prior information (2).

Disability in the short term was rated higher than the long term prognosis of disability. This makes intuitive sense and reflects the natural history of resolution of symptoms. Given that more information on the medical history will assist in making a prognostic judgement, then a difference in judgement between the CON groups and the other two groups is expected. The data on subjects 1 and 5 confirm this position.

However, a difference between the WCB and NWCB groups is found in subjects 3 and 4. It is interesting that both of these men exhibited very different physical signs of LBP. Subject 3 exhibited marked symptomatology whereas subject 4 had minimal objective signs. It is also interesting that differences across groups occurred in these subjects. This may be a reflection of differences in overt signs of LBP.

Subject 3 demonstrated marked symptomatology and the fact that he was not receiving compensation seemed to suggest that his prognosis was better. In fact his present level of disability was rated at 8.3 and this was anticipated to improve to 4.8 in one month, a 35% improvement. In contrast the WCB group rated present disability at 9, and anticipated a 2% improvement to 8.8, in one month. This suggests that patients who are not receiving WCB are expected to demonstrate marked improvement relatively quickly, whereas those receiving WCB are anticipated to change little if at all.

A different scenario was found for subject 4 who demonstrated minor symptoms of LBP. His prognosis was judged to be significantly worse if he was in receipt of WCB. What makes this point more interesting is the fact that for this subject the WCB and NWCB groups did not differ on present disability. A WCB claim was judged to lead to more disability, whereas NWCB involvement led the PTs to expect modest improvement. The long term prognosis evens out across groups for both subjects, with the WCB group expected to fare worse.

It is interesting that the expected benefit from PT reflected a similar trend to that of short term prognosis. There was a significant difference across the group as a whole. But again the differences involved two subjects with differing signs of LBP and scoring trends across the groups. For the subject with mild

LBP, the CON group anticipated less benefit from PT. The same was true for the male subject with mild symptomatology although the difference was not statistically significant. In contrast, the subject with marked symptomatology was anticipated to obtain more benefit from PT, if he was not in receipt of a WCB claim. This was also true for the female subject with marked symptomatology, but the difference was not significant.

All groups scored quite high with respect to the anticipated benefit from PT. The lowest scores were associated with the two subjects with mild symptoms in the CON group. In contrast, the most benefit was anticipated for the two subjects with marked symptoms in the NWCB group. There was an overwhelming affirmative response to the question regarding the need for PT, but it did differ across groups. Sixty-three percent in the CON group responded affirmatively. The score seems high until it is compared to the affirmative response in the other two groups, which was 92% and 79% in the WCB and NWCB groups respectively. The very high affirmative response in the WCB group should be balanced against the prognostic judgement in this group which is lower. It was noted earlier that physicians are thought to overestimate their own effectiveness. The results of this study suggest that this argument could also apply to PTs.

Summary

In summary, it appears from this study that the more objective and simple the measure, the less subject it is to bias and expectancy. In addition, the stronger the visual evidence of LBP, the less likely it is that WCB will have a negative impact in terms of expectancy of outcome. In contrast, there is judged to be a positive effect of no WCB claim, especially when the signs of a back problem are obvious.

It is not clear from this study why the expectancies of outcome differed. It could have been due to the past experience of the PT in their treatment of similar patients. Unfortunately, the memory of past experiences can be biased as negative experiences tend to be more easily remembered. The results may also reflect an unconscious bias. This bias could be against the WCB system or against the patient. Further study is needed to determine the extent of bias amongst PTs. However, taken as a whole the results are reassuring in that although the expectancies of outcome differ according to compensation status. Subjects (patients) are judged on an individual basis, which appears to be driven by their physical signs of LBP, rather than compensation status.

REFERENCES

1. American Medical Association: Guides to the Evaluation of Permanent Impairment. American Medical Association, Milwaukee 1990:96-101.

2. Ashton B, Piper MC, Warren S, Stewin L, Byrne P. Influence of medical history on assessment of at-risk infants. Development med child neurol 1991:33:412-418.

3. Basmajian JV. Research or retrench. The rehabilitation professions challenged. Phys. Ther. 1975:55;6;607-610.

4. Bigos SJ, Battie MC, Spengler DM, et al. A Prospective Study of Work Perceptions and Psychosocial Factors affecting the Report of Back Injury. Spine 1991:16:1;1-6.

5. Carmichael JP. Inter- and intra-rater reliability of palpation for sacro-iliac dysfunction. Journal of Manipulative Physiological Therapeutics 1987:10;4;164-167.

6. Cats-Baril WL, Frymoyer JW. The economics of spinal disorders. In: The Adult Spine: Principles and Practice. Frymoyer JW (Editor-in-Chief) Raven Press, New York. 1991: 85-105

6. Deyo RA, Tsui-Wu YJ. Functional Disability Due to Back Pain. A Population Based Study Indicating the Importance of Socioeconomic Factors. Arthritis and Rheumatism 1987:30;11;1247-1253.

7. French S. Society and the changing nature of illness and disease. In: Physiotherapy a psychosocial approach. French S (Ed.) Butterworth Heinemann, Oxford 1992:1-15.

8. French S. The powerful placebo. In: Physiotherapy a psychosocial approach. French S (ed.) Butterworth Heinemann, Oxford 1992:352-363.

9. Frymoyer JW, Gordon SL. Research Perspectives in Low Back Pain. Report of a 1988 Workshop. Spine 1989:14;12;1384-1389.

10. Haldeman S. Presidential Address, North American Spine Society: Failure of the pathology model to predict back pain. Spine 1990:15;7;718-724.

11. Holbrook TL, Grazier K, Kelsey JL, Staufer RN. The socioeconomic impact of selected musculoskeletal disorders. Chicago, Amer. Acad. Orthop. Surgeons 1984.

12. Illich I. The Epidemics of Modern Medicine. In: Black L et al (eds.) Health and Disease. Milton Keynes, Open University Press, 1984.

13. Krusen EM, Ford DE. Compensation factor in low back injuries. JAMA 1958:166;1128-1133.

14. Leavitt F. The physical exertion factor in compensable work injuries. A hidden flaw in previous research. Spine 1992:17;3;307-310.

15. Magnusson M, Granqvist M, Jonson R, et al. The Loads on the Lumbar Spine during Work at an Assembly Line. The Risks for Fatigue Injuries of Vertebral Bodies. Spine 1990:15; 8;774-779.

16. McKeown T. The role of Medicine, Oxford, Basil Blackwell 1979.

17. Melzack R, Katz J and Jeans ME. The role of compensation in chronic pain: analysis using a new method of scoring the McGill Pain Questionnaire. Pain 1985:23;101-112.

18. Naylor JW. Historical perspective. In: W. Weinstein and S.W. Weisel (Eds) The Lumbar Spine, W.B. Saunders, Philadelphia 1990:1-31.

19. Peat M. Physiotherapy: art or science? Physiotherapy Canada 1981:33;3;170-176.

20. Peters ML, Schmidt AJM, Van den Hout MA, Koopmans R, Sluijter ME. Chronic back pain, acute post-operative pain and the activation of diffuse noxious inhibitory controls (DNIC) Pain 1992:50;177-187.

21. Pither CE, Nicholas MK. The identification of iatrogenic factors in the development of chronic pain syndromes: abnormal treatment behaviour? IN: MR Bond, JE Charlton & CJ Woolf (Eds) Proceedings of the VIth congress on pain. 1991. Elsevier Science Publishers, Netherlands;429-434.

22. Roland M, Morris R. A study of the natural history of back pain. Part I: Development of a reliable and sensitive measure of disability in low-back pain. Spine 1983:8;2;141-144.

23. Rossi U, Pernak J. Low Back Pain: The Facet Syndrome. IN: Lipton S (ed). Advances in Pain Research and Therapy. Vol 13, Raven Press Ltd., New York. 1990:231-244.

24. Simmonds MJ, Kumar S: Pain and the placebo in rehabilitation using TENS and laser. Disability and Rehabilitation (In press).

25. Snook SH, Jensen RC: Cost. IN Pope MH, Frymoyer JW and Andersson G. (eds.) Occupational Low Back Pain. Praeger, New York 1984:115-121.

26. Taylor LT, Twomey JR. Preface. In: Twomey LT and Taylor JR (Eds), Physical Therapy of the Low Back, Edinburgh, Churchill Livingston 1987:xi.

27. Tollison CD. Assessment and treatment at Pain Therapy Centers Programs. Handbook of chronic pain management. Edited by CD Tollison. Williams and Wilkins, Baltimore 1989:656-663.

28. Voudouris NJ, Peck CL, Coleman G. The role of conditioning and verbal expectancy in the placebo response. Pain 1990:43;121-128.

29. Wall PD. The placebo effect: an unpopular topic. Pain 1992:51;1-3.

30. Walsh NE, Dumitru D. Financial compensation and recovery from low back pain. Spine: State of the Art Reviews 1987:2;1;109-121.

CHAPTER FIVE

THE FUNDAMENTAL SKILL OF PALPATION: A REVIEW AND CRITIQUE[4]

Introduction

Palpation is a fundamental assessment and treatment skill for many medical and paramedical professions. In addition, palpation skills are frequently utilised by researchers who test or measure some aspect of human function. Palpation is used in the placement of skin markers for photographic analyses (7) placement of electrodes for EMG analyses (18) and palpation of specific bony landmarks for anthropometric measurements (30) or biomechanical modelling (7).

In the health professions, palpation is a standard component of physical examinations, it provides information on tissue tenderness, temperature, texture, resilience and joint motion (20,21,10,15) the presence of abnormal lumps (35), and the size and position of internal structures. This information is used to formulate diagnoses, plan treatments and assess the efficacy of the treatment.

Accurate and reliable palpation skills are a prerequisite to the acquisition of correct measurements, e.g anthropometric, posture, motion and muscle activity measurements. EMG analysis requires accurate electrode placements in order to optimise the signal, and to ensure that the signal is actually emanating from the correct muscle, crosstalk is minimised. The location of joint lines, bony landmarks or specific muscles are all determined by palpation.

Palpation is a method of measurement. Optimisation of the accuracy of measurement by palpation requires the elimination or control of factors that confound the measurement. The purpose of this review is to identify factors that influence the reliability and validity of palpation findings, specifically as palpation applies to the health care field.

Palpation in Health Care

Palpation is a cheap clinical test, but the sensitivity, the reliability and the validity of the test is controversial. Some practitioners have a strong clinical belief in all tests based on palpation (21). Other practitioners dismiss any test that is based primarily on palpation (24).

Much of the controversy is concerned with the interpretation of the findings. For instance, Maigne and Maigne (21), noted that the oft cited reason for pain over the iliac crest was not due to tenderness of the ilio-lumbar ligament insertion as suggested by Hirschberg et al, (14). A series of 37 dissections showed that the ligament was not even palpable (21).

---

[4]A version of this chapter has been published. Simmonds and Kumar 1993. International Journal of Industrial Ergonomics. 11: 135-143

In addition to the scepticism concerning the interpretation of findings, there is also some doubt whether certain structures or specific motions can be felt (24). Grieve (10) has argued that if sceptics don't feel something, then it is because they don't want to feel something. However, his suggestion that "tireless reiteration" by manual therapists will convince the sceptics, is misguided and misguiding. Obtaining scientific proof of the validity of palpatory tests would be a more appropriate suggestion. At present there is a dearth of palpation literature with sound methodology (13,28).

There are two broad categories of palpatory techniques. One is static palpation and the other is motion palpation (33).

Static Palpation

Static palpation is used to assess the state of the skin, soft tissues, and subcutaneous bony points (10). Temperature, texture and humidity of the skin are noted, as are abnormalities of the patient's sensation. The nature of any swelling, the tone of muscles, the presence of undue tenderness and the relationships between osseous structures are all noted (10,33).

However, the notion that palpation provides objective information (10,16,21) has not been demonstrated. Rather the findings from palpation are based on the subjective perception and interpretation of the examiner. Anecdotal impressions of the accuracy and objectivity of a specific technique are not adequate supporting evidence, especially when there is a vested interest in support of the position. Unfortunately, most texts written by clinical authorities rely primarily on anecdotal impressions (10). It is this reliance on anecdotal evidence that has led to the scepticism of palpatory findings outside of the manipulative professions.

Few studies have addressed the issue of static palpation reliability, primarily due to problems of "blinding" inherent in repeatability studies (5). The use of a sheet to hide skin marks, or the blindfolding of testers has not been useful (5). In addition visual skin cues may be clinically relevant. The removal of skin cues may alter the accuracy of the test and limit the clinical generalisability, albeit Grieve (10) has noted that feeling and looking simultaneously may cause confusion. The variability of the presence of visual cues may also be problematic (26). Miller and collegues reported that the "dimples of Venus" (a visual cue used to locate S2 spinal level) were inconsistently present in size and symmetry and were completly absent in 26% of their subject sample. Intersensory cooperation between vision and touch has not been experimentally addressed in the clinical literature. Research in this area is obviously indicated.

In a different context, Brown (4) investigated the ability to make comparative judgements of the surface roughness of wood. He used lighting conditions which enhanced or diminished visual cues and found that accuracy improved with the simultaneous use of vision and touch. Moreover, lighting conditions which enhanced the visual cues further improved accuracy. The

results of psychophysical studies indicate that touch and vision provide similar levels of performance in isolation, though touch may preempt vision (12). However, the accuracy of perception is improved if both senses are utilised simultaneously (12).

Although intersensory cooperation in palpation has not been addressed specifically. It seems intuitive to suggest that if visual and sensory cues are present, and they are in agreement, then accuracy will be improved. If they are not in agreement, then it suggests that the information provided by palpation may be more reliable.

Anecdotal evidence suggests that clinicians tend to rely on information obtained through touch. This is because clinicians have been observed to close their eyes during palpation testing (22). However, there are no research findings that support this premise. Moreover, apart from palpation of the skin, visual cues will frequently be absent, or at best indirect for deep palpation. The validity and the use of visual cues has not been established but the need is present.

Recent studies on static palpation have utilised a methodology that meets the criteria for 'blinding" whilst not removing any visual cues that may or may not be used (5,34). These studies utilised a pen with a writing fluid that is only visible under ultra-violet light. The findings from both of these studies indicate that the level of reliability varies with the structure palpated.

For instance, Burton et al (5), reported a 5mm error in the location of S2 and L4 spinous processes but this error was doubled to 10mm at the level of T12. Using a slightly different but still blind methodology, Newton and Waddell (29), reported a much greater error, this being 4cm. Only one practitioner participated in each of these studies. The discrepancy in these findings can be explained with reference to the study by Simmonds (34).

Simmonds (34) used 20 subjects in an intra-rater experiment and reported a mean error of 12mm with a range of 0mm - 25mm, in the repeated location of the spinous process. These results show that there is a large range of individual skill in the reproducibility of palpation. The assumption that intra-rater tests are reasonably accurate, is not supported.

A greater level of error is always assumed in inter-rater tests. Burton et al (5) used nine raters and reported a mean 35mm error in inter-rater location of the lumbo-sacral junction. Simmonds (34), used 20 raters and reported a mean 20mm difference in the location of the posterior superior iliac spines (PSIS). The range of error was 7mm to 48mm.

This is a high level of reproducibility error. It is of concern because the PSIS's are key bony landmarks which are used in the assessment of the pelvis, the lower limbs and the spine (10,15,21). Post intervention change in PSIS levels is assessed by palpation, in order to determine the efficacy of the intervention. These test-retest results don't tend to support this practice. If relative bony position as determined by palpation is used, blinded intra-rater assessment is more reliable than inter-rater testing but neither are particularly

accurate. Thus, the level of reliability must be established prior to any investigation that uses palpation for the determination of outcome.

This level of error associated with palpation, helps to explain the low reliability of many clinical tests that are based on palpation. The error in landmark location by palpation may contribute to the low correlations of .68 and .76 obtained between clinical and radiographic spinal range of motion measurements (29).

The high range of intra-rater error in landmark location is one factor that helps to explain the conflicting results of reliability studies using clinical methods of measurement. Other reasons generally relate to differences in methodology and specifically blinding, and to training.

Potter and Rothstein (32) report only 35% agreement between raters measuring PSIS levels. Whilst the results of another study suggest a higher level of agreement (1). They reported ICC values of .88 and greater for tests of inter-rater reliability of several pelvic rotation motions. All motions were measured with an instrument that is placed on the PSIS and anterior superior iliac spine (ASIS) (1). The high correlations suggest that there was a high level of agreement but that doesn't necessarily mean that they were accurate in landmark location. However, although testers palpated and placed skin markers, it is not clear whether the skin markers were left in position for the next tester. The specific error associated with skin marking was not reported. The investigators noted that skin markers were moved but the distance between markers was not measured. Finally, a training period was utilised in this study. This allowed subjects to become familiar with the instrument, and practice the procedure. The discrepancy between this and the previous study may be explained by the lack of blinding and the effect of training.

This makes intuitive sense and studies utilising training programmes for testers prior to the investigation have reported higher levels of reliability (1,6), than those which have not specifically trained the testers (32). The latter approach obtains poorer levels of reliability, but it probably gives a clearer indication of actual clinical practice.

The learning effect of clinical experience is not clear. Anecdotal evidence suggests that clinical experience improves palpation techniques (10). However, experimental evidence of this assertion is lacking. In fact, Carmichael (6), reported a wide range of palpation abilities in both inexperienced and experienced chiropractors. Moreover, the work of Brown (4) has shown that there is no significant difference in the sensory discriminitive ability of skilled and unskilled workers. This implies that differences -if present - are a result of interpretation rather than sensation.

The development of idiosyncracies that occur with clinical experience may be one factor that predisposes to poor reliability. This may be more evident in motion rather than static palpation.

In research, where accuracy of measurements is vital, training helps to determine that the learning effect of measurement has stabilised prior to data

collection. The learning effect of goniometric measurement has been shown by (9).

Fundamental to goniometry is the requirement of location by palpation of the bony landmarks specific to the joint to be tested. The accuracy of landmark location as well as training will influence the accuracy of the measurement.

Boone and collegues illustrated this particular point in a reliability study (3). They showed that the reliability of goniometric measures varies with the specific joint tested. Lower limb joints are associated with less reliability than upper limb joints.

For example, the reliability associated with the hip was .55 compared to .97 for the shoulder. This occurred despite the fact that the shoulder has a complex motion due to the mobility of the pectoral girdle. However, the shoulder joint is more superficial than the hip. The arm is also lighter, smaller and easier to handle than the leg. This makes location of bony landmarks for the shoulder joint much easier to identify. This fact and the relative ease of handling a limb probably contributes to the reliability of measurement.

It makes intuitive sense that palpation of superficial structures is more accurate than that of deep structures. Bloom and collegues quantified these differences in a psychophysical study aimed to identify the stimulus dimensions that determine the detection of simulated breast lesions (35). Steel chunks of various dimensions were embedded in silicone breast models at various depths. Five subjects palpated these models in a lump detection task. Both the size and the depth of the lump influenced the detection rate. Also, fixed lumps had a higher detection rate than mobile lumps. These factors may have influenced the results of a clinical study on the detection of breast lesions, and axillary nodes (35). The interobserver variation in the clinical assessment of patients with breast lesions was large for many of the examination items that depended on palpation. There was particularly poor agreement (45%) in recording the presence or absence of axillary nodes and in sizing the primary lesion (55%). The variation in sizing of the primary lesion may be reflective of differences in the ability to judge the size, rather than to feel the size of the lesion. However, the results of this study are not reassuring given the consequences of not detecting breast lesions.

The lack of precision of clinical measures was examined in a study by Miller et al (26). They examined the Schober technique of lumbar flexion using blind methodology. The Schober technique involves marking the skin at the level of S2 drawing a 10 cm line in a proximal direction and a 5 cm line in a distal direction. The line is measured with the subject standing erect and then remeasuring the length of this line as the subject flexes.

Good reliabilities have been reported when there is no blinding of testers (25). However, low reliabilities occur when blinding of skin marks was used in the study by Miller et al (26). A number of factors which contributed to the low reliability were reported. Amongst these was the problem of the variability in the presence of visual cues to locate landmarks.

Summary

In summary, the reliability of static palapation is extremely variable. The level of reliability varies with; the rater -intra- vs inter-rater, the structure -upper limb vs lower limb, the definition of the structure, the degree of training and the standardization of the technique.

Intra-rater reliability is greater than inter-rater reliability. This suggests that each examiner develops their own criteria for location. However there is a wide range of skill in the reproducibility of static palpation, and this has little to do with experience. Inter-rater reliability is poor but improves with standardisation and practice. It is important that the level of reliability be determined prior to any experimental study. The validity of research data may be compromised because of errors in location by palpation. There is presently a need for systematic evaluation of each of the factors that affect the reliability of static palpation.

Finally, there is a need to validate static palpation. This is especially important for key osseous landmarks. These landmarks are not only evaluated for their own relative position, but also provide starting points from which to palpate other more difficult structures. Location error associated with the secondary structure is compounded when there is error in the location of key landmarks.

Motion Palpation

All of the issues relating to the reliability of static palpation apply to motion palpation. However, there are additional issues. There is still the problem of accurate location of the joint to be tested. In addition there are problems associated with accurate application of a force, either an external force applied by the examiner, or an internal force applied by the patient's muscles. The examiner is required to perceive and make judgements about various qualities of motion or fixation of joints, and tissue compliance. Discussion of the validity of the various tests is beyond the scope of this paper. Rather a discussion of the factors influencing reliability of motion palpation will be presented.

Motion palpation is subdivided into passive and active palpation tests. Passive motion palpation attempts to assess the quality of motion between adjacent articular segments (30). This is done with the examiner moving the joint or body segment in question. The examiner then perceives the magnitude of motion, the ease or resistence to motion and the quality of the "end-feel" of motion.

Active motion palpation is performed with the subject actively moving the joint. The examiner feels for the change in relationship between specific osseous points (28). Beal (2,33) (Cited by Russel, 1983) has stated that motion sense is the culmination of palpatory skills and is the limiting factor in the art of manipulation (2).

Despite extensive clinical use many procedures are not well described or recorded in clinical trials (28). There are some descriptions of certain techniques in textbooks, but these are qualitative descriptions with no mention of the magnitude or rate of applied force. Terms such as "gentle distraction" or "firm pressure" are open to individual interpretation. The lack of standardization is compounded as these skills are traditionally taught by "clinical experts" who may superimpose idiosyncracy on the technique.

Most investigations have examined the axial spine and the pelvis. The rationale for testing intervertebral or sacro-iliac joint motion rests on the premise that 1). abnormal movement causes pain and/or dysfunction and 2). abnormality in motion can be clinically detected. Given that this premise is correct, and ignoring for the present, the specificity and sensitivity issues of motion testing, it is necessary to consider some factors that influence the application and therefore the results of the tests.

The accuracy of location of the structure under test was discussed previously. The error associated with accurate location may be a major factor in the poor reliability of motion tests (11,17,28). It is not clear how the examiners identify the intervertebral segment which is "fixated" or hypermobile. Verbal identification during palpation, e.g "this segment is stiff" is merely a test of motion palpation. However, if the level of fixation is determined only by palpation, this is a test of the accuracy of location by palpation as well as motion perception. Thus reliability will be lower as there is the potential for error in each aspect of the test.

One of the problems in testing reliability is that as testing of musculoskeletal tissues is carried out, the tissues may in fact change (16). This may confound reliability and validity investigations. The use of manufactured models provides a useful method that may avoid this problem.

Harvey and Byfield (11) utilised a cadaveric vertebral spine which they modified with screws and bushings to control specific segmental motion. The model was covered with a chamois leather in order to eliminate visual cues. Examiners were required to determine the presence or absence of fixation at each lumbar and lumbosacral level. The observed rate of correct motion palpation was fairly high, but so was the rate expected by chance, 81% vs 71% respectively. The authors noted that although specificity was quite high the sensitivity was low, indicating a failure to identify the fixation.

The amount of motion allowed at each segment was not reported. So neither the sensitivity of the model, nor that of the examiners can be determined. During testing fixation was present at only one level. Neither gradations of motion, nor multiple fixations were tested for their identification.

The use of mechanical models to examine the sensory aspects of palpation is a positive step. Accurate and quantitative psychophysical measurements can be obtained. The physical components of the external forces applied by the examiner can be quantified. In addition, alteration of the

internal stresses of the model will allow the perceptual abilities of the examiners to be measured. Evans (8), has attempted this using a device designed to simulate resistance to motion. Unfortunately, nonlinearity problems of the device make any conclusions from its use suspect. It can be argued that biological tissue does not react to mechanical stress in a linear manner. However, there is no evidence to suggest that the nonlinearity of the device matched that of biological tissue. Further study utilising models is warranted, and validation of the models is a necessary step. A model that incorporates physiological and biomechanical properties of human tissue is yet to be developed.

Quantification of the perceptual abilities of the examiners will determine the actual objectivity of palpation findings. Palpation findings are usually discussed and recorded as objective tests (10,16,21). Information that doesn't fit the clinical theory is ignored or disbelieved (10). For example, Lee (21), acknowledges the anatomical, biomechanical, and radiological evidence that largely precludes movement in the sacroiliac joint, but writes that the presence or absence of sacroiliac joint mobility and its significance to the patients complaints are best judged by accurate, objective, clinical evaluation (21).

The examiner's judgement of a clinical test is accepted as providing accurate objective evidence. At the same time the subjective nature of the patient's response is deemed to be of lesser reliability (16). Yet the only tests that have obtained a reasonable level of reliability, are those that require the patient's response. Potter and Rothstein (32) examined the inter-rater reliability of 13 tests for the sacro-iliac joint. A reasonable level of reliability (greater than 70% agreement) was obtained on only 2 tests. These two tests relied solely on patient response and provided no information on sacroiliac position or motion (32).

A similar result was reported by Keating et al, (17). These authors examined the inter-rater reliability of eight tests for lumbar segmental abnormality. The two tests for osseous and soft tissue pain were the most reliable. They reported Kappa values of .48 and .30 respectively for each pain test. They also reported mean Kappa values of .09 for passive motion palpation, .07 for muscle tension palpation, .09 for active motion palpation and .00 for misalignment palpation. The guidelines suggested for judging the strength of Kappa coefficients were:- $0 < K < .4$ = marginal reproducibility, $.4 < K < .75$ = good reproducibility, $K \geq .75$ = excellent reproducibility.

This suggests that palpation tests are open to interpretation and should not be regarded as objective measures. There is a definate need for standardization of technique in order to improve these poor results.

It is unlikely that reliability will improve unless there is improved standardization of technique. It also seems necessary to periodically "recalibrate" the technique. Anecdotal evidence suggests that experience is an asset (21,10). Whilst experimental evidence has shown that experience has no

effect (31), or that it contributes to lower levels of both intra- (13) inter-rater agreement (27).

The use of a model on which to train and to test examiners is highly recommended. Reliability is not likely to improve unless techniques are standardised. The use of unreliable measures on which to base a diagnosis and treatment serves no useful purpose except to support the arguments of the sceptics.

Standardization of technique and training according to the set standards, may be one way in which to avoid the systematic examiner bias which is found at present. For instance, Matyas and Bach (22) used a force platform and biomechanical algorithm to measure the specific application of force applied by therapists. The therapists were required to report the specific point at which they detected resistence to motion. The mean intertherapist correlation was only .22 (22). A systematic bias in the magnitude of applied force was found between therapists and this may explain the poor inter-rater reliability in the detection of joint motion or tissue compliance.

Systematic difference in force application was also suggested to be a factor leading to low reliability of diagnostic tests for the knee joint (23). The authors tested the reliability of clinical judgements of ligament integrity in the knee. A systematic difference between therapists was reported. Therapists who reported joint laxity also reported the occurrence of pain during the test.

This suggests that therapists who use a greater magnitude of force, will report the presence of pain and abnormal motion more frequently. However, it is possible that the rate and direction of force will also affect the result in terms of motion, and perception of tissue compliance. Neither of these factors have been addressed.

Summary

The lack of standardization, poor reliability and questionable validity of many motion palpation tests is a serious problem. Motion palpation forms the basis for diagnosis and treatment. The efficacy of spinal manipulation/mobilization has only limited empirical support (31). It is necessary to determine the factors that limit the response to treatment.

It is possible that the lack of reliability of palpatory techniques for diagnosis and treatment, contributes to the limitation of effect. This does not mean that clinical tests should be dismissed. Rather it is necessary to objectively evaluate the techniques involved in clinical tests in order to determine their validity.

Valid clinical tests are of little use if they are unreliable, therefore quantitative standardization of testing and training is imperative. The use of manufactured models is recommended. Models can be used to measure the perceptual abilities of the examiner, and to quantitify parameters of the forces applied by the examiner during specific tests.

Finally, standardised and valid tests are of no use if they are applied in the wrong place. Systematic, blind examination of the factors that influence the reliability and validity of location palpation is indicated. This will help to formulate a model which can predict the reliability of specific measurements or tests based on palpation. This information is important to clinicians and researchers alike.

REFERENCES

1. Alviso DJ, Dong GT and Lentell GL. Intertester reliability for measuring pelvic tilt in standing. Physical Therapy 1988:68;1347-1351.

2. Beal MC. Motion sense. Journal of the American Osteopathic Association 1953:53;151-153.

3. Boone DC, Azen SP, Lin C-M, Spence C, Baron C, and Lee L. Reliability of goniometric measurements. Physical Therapy 1978:58;1355-1360.

4. Brown ID. Visual and tactual judgements of surface roughness. Ergonomics 1960:3;51-61.

5. Burton K, Edwards VA, and Sykes DA. "Invisible" skin marking for testing palpatory reliability. Journal of Manual Medicine 1990:5;27-29.

6. Carmichael. Inter- and intra-examiner reliability of palpation for sacroiliac dysfunction. Journal of Manipulative Physiological Therapeutics 1987:10;164-17.

7. Cheng C and Kumar S. A three-dimensional static torso model for the six human lumbar joints. International Journal of Industrial Ergonomics 1991:7;327-329.

8. Evans DH. The reliability of assessment parameters: accuracy and palpation technique. In: Grieve G. (ed.) Modern Manual Therapy. Churchill Livingston Edinburgh 1986:498-502.

9. Goodwin J, Cark C, Deakes J, Burdon D, and Lawrence C. Clinical methods of goniometry: a comparative study. Disability and Rehabilitation 1992:14;10-15.

10. Grieve GP. Common Vertebral Joint Problems Edinburgh: Churchill Livingstone 1981.

11. Harvey D and Byfield D. Preliminary studies with a mechanical model for the evaluation of spinal motion palpation. Clinical Biomechanics 1991:6;79-82.

12. Heller MA. Visual and tactual texture perception: Intersensory cooperation. Perception and Psychophysics 1982:31;339-344.

13. Herzog W, Read LJ, Conway PJW, Shaw LD and McEwen MC. Reliability of motion palpation procedures to detect sacroiliac joint fixations Journal of Manipulative Physiological Therapeutics 1989:12;86-92.

14. Hirschberg GG, Froetscher L and Naeim F. Iliolumbar syndrome as a common cause of low back pain: diagnosis and prognosis. Archives of Physical Medicine and Rehabilitation 1979:60;415-419.

15. Hoppenfield S. Physical Examination of the Spine and Extremities. Appleton-Century-Crofts. New York 1976.

16. Johnston WL. The role of static and motion palpation in structural diagnosis. In: Workshop on the Research Status of Spinal Manipulative Therapy. Bethesda Maryland. 1976:249-253.

17. Keating JC, Bergmann TF, Jacobs GE, Finer BA and Larson K. Interexaminer reliability of eight evaluative dimensions of lumbar segmental abnormality Journal of Manipulative Physiological Therapeutics 1990:13;463-470.

18. Klein AB, Snyder-Mackler L, Roy SH, and DeLuca CJ. Comparison of spinal mobility and isometric trunk extensor forces with electromyographic spectral analysis in identifying low back pain. Physical Therapy 1991:71;445-454.

19. Lee D. The Pelvic Girdle Churchill Livingstone Edinburgh 1989:15-91.

20. Lewit K. Manipulative Therapy in Rehabilitation of the Locomotor System Butterworths London 1985:109.

21. Maigne J-Y and Maigne R. Trigger point of the posterior iliac crest: Painful iliolumbar ligament insertion or cutaneous dorsal ramus pain? An anatomic study. Arch Phys Med Rehabilitation 1991:72;734-737.

22. Matyas TA and Bach TM. The reliability of selected techniques in clinical arthrometrics. The Australian Journal of Physiotherapy 1985:31;175-199.

23. McClure PW, Rothstein JM and Riddle DL. Intertester reliability of clinical judgements of medial knee ligament integrity. Physical Therapy 1989:69;268-275.

24. McKenzie RA. Mechanical diagnosis and therapy for low back pain: toward a better understanding. In: Twomey LT and Taylor JR (eds.)

Physical Therapy of the Low Back. Churchill Livingstone New York. 1987:157-173.

25. Merritt JL, McLean TJ, Erickson RP and Offord KP. Measurement of trunk flexibility in normal subjects: Reproducibility of three clinical methods. Mayo Clinic Proc. 1986:61;192-197.

26. Miller SA, Mayer T, Cox R and Gatchel RJ. Reliability problems associated with the modified schober technique for true lumbar flexion measurement. Spine 1992:17;345-348.

27. Mior SA, McGregor M, Schut B. The role of experience in clinical accuracy.Journal of Manipulative Physiological Therapeutics 1990:13;68-71

28. Mootz RD, Keating JC, Kontz HP, Milus TB and Jacobs GE. Intra-and interobserver reliability of passive motion palpation of the spine. Journal of Manipulative Physiological Therapeutics 1989:12;440-445.

29. Newton M, Waddell G. Reliability and validity of clinical measurement of the lumbar spine in patients with low back pain. Physiotherapy 1991:77;796-800.

30. Nowak E. Practical application of anthropometric research in rehabilitation. International Journal of Industrial Ergonomics 1992:9;109-115.

31. Ottenbacher K, DiFabio RP. Efficacy of spinal manipulation/mobilization therapy. A meta-analysis. Spine 1985:10;833-837.

32. Potter NA and Rothstein JM. Intertester reliability for selected clinical tests of the sacroiliac joint. Physical Therapy 1985:65;1671-1675.

33. Russell R. Diagnostic palpation of the spine: A review of procedures and assessment of their reliability. Journal of Manipulative and Physiological Therapeutics 1983:6;181 - 183.

34. Simmonds MJ. The reliability of palpation skills in the therapeutic professions. In: Kumar S (ed.) Advances in Industrial Ergonomics and Safety IV. Taylor and Francis London 1993:665-671.

35. Yorkshire Breast Cancer Group. Observer variation in recording clinical data from women presenting with breast lesions. British Medical Journal 1977:2;1196 - 1199.

CHAPTER SIX

LOCATION OF BODY STRUCTURES BY PALPATION: An experimental study[5]

Introduction

Palpation is the process of physical examination of the body by means of touch. It is a fundamental skill that has long been practised by medical practitioners (7). However it is a skill that is not unique to medical practitioners. Any profession that measures anthropometric characteristics for motion analysis task analysis or for experimental study uses palpation. As a standard component of musculoskeletal examinations palpation provides important information about tissue tenderness, temperature, texture, resilience, and joint motion (10,9,5,6). Palpation also provides information about the presence of abnormal lumps (15) and the size and position of internal structures. The information obtained from palpation is used to formulate diagnoses and plan treatments.

Given the fundamental importance of palpation in clinical and research applications it is surprising that the skill has not received more scrutiny.

For many years the results of palpation testing were accepted as providing accurate and objective information (5,9). For many therapists this remains the case despite contradictory experimental evidence (14). Strong and unquestioning belief in clinical tests and theories may be due to the fact that complex clinical theory has developed prior to a sound scientific and verifiable base (11). Moreover clinical theories are frequently reiterated and become the accepted dogma (1). Experimental evidence does not generally lend support to this clinical view.

There is little literature that specifically reports the palpation accuracy in the human body. The accuracy of location of bony landmarks was investigated by Burton (2). He used an invisible marking pen and measured the distance between consecutive marks for spinal levels S2, L4 and T12. The distance between consecutive marks varied between location and for within vs. between raters. The mean intra-rater distances were 5mm for S2 and L4 landmarks and 10mm for the T12 landmark. The mean inter-rater distance was reported to be 35mm for the T12 landmark. These results suggest a reasonable level of accuracy especially for one rater.

Palpation skills are also important to ergonomists. The accuracy of anthropometric data posture measurement task and motion analysis a determination of link lengths is dependent on the accurate location and placement of body markers. EMG analysis requires accurate electrode

---

[5]A version of this chapter has been published. Simmonds and Kumar 1993. International Journal of Industrial Ergonomics 11: 145-151

placements in order to optimise the signal and to ensure that the signal is actually emanating from the correct muscle and is free of crosstalk. The location of joint lines bony landmarks or specific muscles is determined by palpation.

The purpose of this study was to investigate how the physical characteristics of a structure influence the amount of error associated with its location by palpation.

METHOD

Sample

Subjects for this study were volunteer physical therapists and students in the final year of a physical therapy programme (PT's). A total of 20 PTs participated in the experiment. All were in good physical health with normal cognitive capability. The subject sample represented a range of body types. The height of the subjects ranged between 5 to 5.6 feet for females and 5.25 to 6 feet for males. The age range of the subjects was 24 to 41 years. All subjects had experience in palpating the landmarks used in this study.

Experimental design

A test-retest experimental design was utilised for this intra- and inter-rater reliability study.

Tasks

The task of the therapist was to palpate and mark specific anatomical structures on the subject using a pen with invisible ink. The structures were: a). the anterior border of the lateral ligament of the knee at the level of the knee joint (KNEE); b). the spinous process of L4 (SPL4); c). the posterior superior iliac spine (PSIS) and d); the transverse process of L4 (TPL4).

Testing postures

The testing postures were standardised in the following manner. Palpation of KNEE was conducted with the subject seated on the side of the plinth. The hip on the palpated side was flexed abducted and laterally rotated. The knee was medially and laterally rotated during the palpation procedure in order to better identify and mark the lateral ligament.

All other landmarks were palpated and marked with the subject lying prone. A pillow was placed under the abdomen to improve subject comfort and because this is standard clinical practice.

Procedure

Random assignment of 20 therapist/subject partnerships was conducted. The therapists were instructed to palpate each of the four anatomical structures. They were asked to mark each structure with a dot using a pen with invisible

ink. Five minutes after this marking the skin of the subjects was examined by the principal investigator. If there was no erythema or other visible cue remaining the therapist palpated and remarked the skin of the subject. The subjects were requested not to provide any verbal or other cue to the therapist which could influence the palpation technique of the therapist.

The same palpation procedure was followed with the second subject. However therapists were requested to mark the skin with a small cross. This allowed for the measurement of inter-rater distances. Two subjects were evaluated by each therapist.

After the identification marking was finished the invisible marks were illuminated with the ultra-violet light. The now visible "dots and crosses" were marked with a skin crayon. The distances between these marks was then measured with a flexible plastic ruler.

Equipment

The pen used in this experiment was a Sanfords security marker. A Blak-Ray UVL 21 (Ultraviolet Products Inc. San Gabriel California) was used to illuminate the marks. Both instruments are illustrated in Figure 6.1.

Analysis

Descriptive and inferential statistics were computed. The main analysis was a repeated measures MANOVA with both location (4 levels) and occasion 1 and 2 as repeated factors. Post-hoc analysis was conducted using paired t-tests. The alpha level of $p < 0.05$ was accepted as significant for all tests.

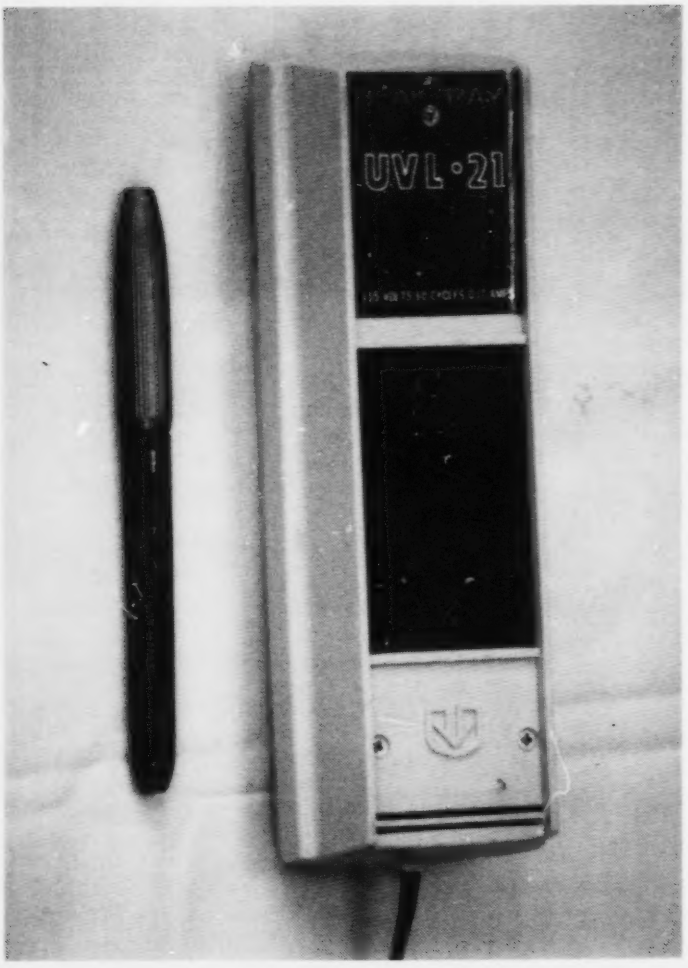


Figure 6.1 Marking pen and ultraviolet light

RESULTS

The descriptive data on the intra-rater distances for each body location are presented in Table 6.1. The shortest distances between two marks indicating the greatest degree of reproducibility occurred at the KNEE and PSIS. The spinous process of L4 and transverse process of L4 were both associated with greater distances between consecutive marks. A minimum distance of zero occurred at each location indicating perfect reproducibility on some tests. In contrast the maximum distance between tests showed a great deal of variability and was dependent on location. The range of test distances are reflected in the standard deviations. These values are smallest for PSIS and highest for the transverse process.

The descriptive data for the inter-rater tests is presented in Table 6.2. The distance between inter-rater tests is greater at every location. However the pattern of results is slightly different. Again the KNEE and TPL4 were respectively associated with the greatest and least level of reproducibility. The mean distance for the KNEE was 12mm with a standard deviation of 4mm. With the exception of TPL4 the minimum distance for each test is greater for inter- vs. intra-raters. The maximum inter-rater distances are greater than intra-rater distances across all locations.

The MANOVA was significant (Pillai =.984 p =.0001). For the intra-rater test the results of the post hoc analyses showed that KNEE and SPL4 were significantly different from each other and both SPL4 and TPL4 were significantly different from PSIS. Inter-rater differences in location were revealed between KNEE and all other locations and between SPL4 and TPL4. Table 6.3 presents the results of the post hoc analysis between intra-and inter-raters. There were significant differences between intra-and inter-rater means of the distances for all locations except SPL4.

Figure 6.2 Illustrates the illumination of marks with the light

Figure 6.3 Illustrates the illumination of crosses made in the inter-rater test.

There was a large range of individual variation in reproducibility skill. The mean intra-therapist distances averaged over location ranged between 9 and 20mm. The minimum distance also showed a large variation being 0 to 10mm. The maximum distance ranged from 0 to 48mm.

Table 6.1. Descriptive statistics of distances between consecutive tests for intra-rater experiment (n=20).

Location	Mean (mm)	S.D.	Coeff of var.	Minimum	Maximum
Lateral ligament of the knee	8	6	.75	0	18
Posterior superior iliac spine	8	5	.63	0	17
Spinous process L4	12	7	.28	0	25
Transverse process L4	14	11	.78	0	37

Table 6.2. Descriptive statistics of distances between consecutive tests for inter-rater experiment (n=20).

Location	Mean (mm)	S.D.	Coeff of .var	Minimum	Maximum
Lateral ligament of the knee	12	4	.33	7	19
Posterior superior iliac spine	20	13	.65	7	48
Spinous process L4	16	8	.50	8	35
Transverse process L4	25	12	.48	0	43

Table 6.3. Results of post-hoc t-tests between intra- and inter-rater distances for each location (n=20).

Location	Intra-rater mean (mm)	Inter-rater mean (mm)	t-value (prob.)
Lateral ligament of the knee	8	12	-2.98 (.008)
Posterior superior iliac spine	8	20	-4.33 (.0001)
Spinous process L4	12	16	-1.46 (N.S)
Transverse process L4	14	25	-2.47 (.023)

N.S. = Non-significant
Significance level $p < .05$)

DISCUSSION

The results of this study confirm that there is a significant amount of error associated with palpatory findings. The results also indicate that there is variability in the magnitude of error which is related not only to the palpation skill of the individual or individuals but also to the structure being palpated.

The lowest level of error was found at the knee both within and between raters. This indicates that the line of the ligament was fairly easy to distinguish from the underlying tissue. The fact that both the ligament and the joint line are superficial and are generally quite distinct lines contributes to the lower rate of error associated with the identification of this point.

The PSIS was associated with low intra- but high inter-rater error. This is probably explained by the fact that the although the PSIS is superficial it is sometimes an indistinct bone and frequently assymetrical. Each therapist uses their own individual reference point for its identification.

The amount of error associated with the PSIS is of some concern because of its status as a key landmark. Whilst it is acknowledged that the presence of pelvic assymetry is common and frequently unrelated to any pathology. Nevertheless pelvic symmetry is frequently tested clinically and treatment decisions are based on the results of the tests. In addition changes in pelvic symmetry are used as outcome measures in the assessment of treatment efficacy (4). It is clear that conclusions based on palpation evidence are suspect if there is more than one rater. This may also explain the poor reliability of clinical tests of the pelvis and sacro-iliac joint (13).

The spinous process of L4, -like the PSIS, is a superficial bony structure. However, it is smaller in size than the PSIS. Moreover, identification of the L4 spinous process is based on a secondary level of palpation. Judgement of specific spinal levels is made with reference to another bony landmark with which it is assumed to have a fairly consistent relationship. In the case of SPL4 the primary landmark is usually the PSIS or the iliac crest. The PSIS is considered to be at the S2 spinal level whereas a horizontal line drawn at the level of the iliac crest is considered to be at the junction of L4/5. Given these "definitive levels" the palpator counts the bony prominences in a proximal direction until L4 is reached.

The error associated with the identification of SPL4 tended to be due to error in the identification of the correct spinal level. This of course may be reflective of the error associated with the identification of PSIS and compounded at L4. There was very little discrepancy in horizontal distances indicating that spinal processes were palpated.

The results from this study resolve the conflicting results between that of Burton et al (2) and Newton and Wadell (12). Burton and collegues reported a 5mm distance between consecutive skin marks on the spinous process of L4 whereas Newton reported a 4cm difference. The present study found a mean distance of 12mm and a range between 0 and 25mm. Both Burton and

Newton tested the reliability of only one rater and their results are an indication of the intra-individual range. The magnitude of this range highlights the requirment to conduct reliability tests prior to collecting measurements which rely on palpation. This is particularly important when precise and sensitive measurements are needed.

The precise identification of spinal level is necessary for any localised therapeutic or diagnostic test. It is possible that errors in the technique of palpation may contribute to difficulties associated with the diagnosis and management of low back pain.

Precise identification of spinal level is frequently implied in ergonomic research especially in EMG studies. Tests of the reliability and validity of identification may well be carried out but they are not usually reported. It may be argued that the statistically significant level of error associated with SPL4 is not clinically significant. However, Krag and collegues (8) have recently reported that there is an intersegmental difference in the activity level of erector spinae and multifidus. These authors used X-ray validation of spinal level and wire electrodes to measure the EMG signal during loaded and unloaded symmetric and assymetric activities. This means that precise identification of spinal level is important in EMG analyses.

Precise identification of spinal level is also a prerequisite for the accurate calculations of biomechanical stresses. Cheng and Kumar (13) used a three-dimensional static torso model and showed that there was a compression force difference of approximately 15 newtons between each spinal level.

A clinically significant level of error varies with the sensitivity of the test. Moreover relevance of the error is dependent on the specificity of treatment effect. The accurate identification of a specific spinal level is hardly relevant if the treatment is at a general level.

The transverse process of L4 is a deeply placed fairly small bony structure. It is a key point of palpation used by some clinicians in their determination of vertebral position. The transverse process like the spinous process is also a secondary point of palpation.

The error associated with TPL4 was present in the horizontal as well as vertical direction. This indicates that there was not only error in vertebral level but also in the identification of the transverse process. It is possible that facet joints were identified as transverse processes in some instances. It seems intuitive that any clinical assertions of vertebral position based on the position of the transverse process must be questioned. But given the lack of symmetry in the normal assymptomatic spine the validity of these diagnoses is questionable anyway.

CONCLUSION

The variable level of reliability and the lack of proven validity of such a fundamental skill is cause for concern especially when palpatory findings

provide the major bases for diagnoses treatment and quantitative analyses. This study provides quantitative evidence of the error associated with palpation. Moreover it shows that there is a systematic error associated with specific body landmarks and with different palpators. The reportedly poor reliability of many clinical tests may be due to errors associated with palpation.

REFERENCES

1. Bogduk N and Twomey LT.Clinical Anatomy of the Lumbar Spine Churchill Livingston London 1987.

2. Burton K. Edwards VA, Sykes DA. "Invisible" skin marking for testing palpatory reliability. Journal of Manual Medicine 1990:5;27-29.

3. Cheng C, Kumar S. A three-dimensional static torso model for the six human lumbar joints. International Journal of Industrial Ergonomics 1991: 7;327-339.

4. Cottingham JT, Porges SW and Richmond K. Shifts in pelvic inclination angle and parasympathetic tone produced by rolfing soft tissue manipulation. Physical Therapy 1988:68;9;1364-1370.

5. Grieve GP. Common Vertebral Joint Problems. Edinburgh: Churchill Livingstone. 1981.

6. Hoppenfield S. Physical Examination of the Spine and Extremities. Appleton-Century-Crofts. New York 1976.

7. Katz D. A sense of touch. The technique of percussion palpation and massage. The British Journal of Physical Medicine 1936:(Dec);146-148.

8. Krag MH, Fox H, Alaranta H, Pope MH and Chattopadhyay S. EMG activity of selected components of the erector spinae and multifidus muscles in the lumbar spine measured using wire electrodes during the performance of loaded and unloaded tasks. In: Kumar S. (ed.) Advances in Industrial Ergonomics and Safety IV. Taylor and Francis London 1992:1463-1467.

9. Lee D. The Pelvic Girdle. Churchill Livingstone Edinburgh 1989:15-91.

10. Lewit K. Manipulative Therapy in Rehabilitation of the Locomotor System London: Butterworths 1985:109.

11. Matyas TA and Bach TM. The reliability of selected techniques in clinical arthrometrics. The Australian Journal of Physiotherapy 1985:31;5;175 - 199.

12. Newton M, Waddell G. Reliability and validity of clinical measurement of the lumbar spine in patients with low back pain. Physiotherapy 1991:77;12;796-800.

13. Potter NA and Rothstein JM. Intertester reliability for selected clinical tests of the sacroiliac joint. Physical Therapy 1985:65;11;1671-1675.

14. Twomey LT. Editorial. The Australian Journal of Physiotherapy 1985:31;174.

15. Yorkshire Breast Cancer Group. Observer variation in recording clinical data from women presenting with breast lesions. British Medical Journal 1977:2;1196-1199.

CHAPTER SEVEN

PAIN AND THE PLACEBO IN REHABILITATION[6]

Introduction

Clinicians in the healing arts have long utilised the placebo effect. They have been used benevolently by physicians and other health professionals in order to reduce symptoms and they are successful. In part the placebo effect is evidence of the power of "the encounter" between patient and health professional. It is an example of the art of medicine. The denigration of the placebo is associated with the elevation of the science of medicine and a belief in the omnipotence of this science (51). In this context the placebo was used as a simple but simplistic check on the veracity of organic symptoms. A positive placebo reaction was interpreted as proof of a somatic hallucination (19).

A more balanced view has now developed. There is a recognition of the complexity of the placebo effect and a greater acceptance of the mind/body duality. Greater acceptance of the placebo has also occurred as a result of the evidence that physiological responses result from the administration of "inert" substances (45,28). All treatments comprise physiological and psychological effects. The magnitude of each effect will depend on a multitude of factors. These include but are not limited to; the pathophysiology the pathopsychology the previous experiences of the patient and the skills and previous experiences of the health professional (67), and to social modelling. The physiological and psychological effects due to the placebo response are often termed non-specific effects (52).

The placebo effect has been demonstrated in numerous studies but it is not well understood theoretically (60). It may be due to the effects of classical conditioning or expectancies (60). Expectancies attitudes and the behaviours of both patient and clinician have all been shown to play a role (70,3,22,63). Past experiences of the patient will influence their expectancies and beliefs but also contribute to classical conditioning. Essentially conditioning and expectancy paradigms are not mutually exclusive and there is probably a cognitive or expectancy component in classical conditioning (63). For example, in drug trials placebo effects are stronger when the placebo is given after the true medication (36). Also the time course of saline injections strongly mimics that of morphine in morphine experienced patients (63). It is this phenomenon that has led to the investigation of the role of opiates in the placebo response.

This role seems reasonable. Rats can be classically conditioned to footshock analgesia which can be reversed by the opiate antagonist naloxone

---

[6]A version of this chapter has been accepted for publication. Simmonds and Kumar 1993. Disability and Rehabilitation In press.

(45). A key problem with investigating the mechanisms of placebo analgesia is that noxious tests involve stress. Stress can and does induce analgesia. Stress increases beta-endorphin levels in plasma and cerebrospinal fluid (45).

The "positive" role of stress appears to contradict Evans's (16) hypothesis that administration of an "analgesic" reduces stress and anxiety and it is this reduction of stress that leads to placebo induced analgesia. Evans's hypothesis has not actually been validated (66). However it is interesting to speculate whether there is an optimum level of stress and anxiety.

Support for this speculation is offered by Tierney et al (57). Tierney and colleagues showed that different levels of stress activated different analgesic systems. In mice short swims induced non-opioid analgesia whereas longer swims activated opioid analgesia. Moreover, activation of the opioid systems appeared to suppress the expression of non-opioid analgesia.

Al Absi and Rokke (2) investigated the role of stress and anxiety in humans. They not only induced different levels of anxiety in their subjects but also made the anxiety condition relevant or irrelevant to the test situation. In this study they used the cold pressor to induce pain. It was found that the high anxiety group reported higher levels of pain compared to the low anxiety group. However if subjects were given information that was irrelevant to the test situation i.e. they were given high anxiety inducing information about electric shocks but were not exposed to them then the group reported low levels of pain (2). This may be due to the distraction effect of irrelevant anxiety. However the results also suggest that anxiety should not be considered a homogenous factor with similar effects in all situations. Both the amount of stress and the cause and focus of anxiety will influence pain perception and physiological responses.

It is intuitive that the amount of stress influences the placebo response. But the relationships between stress and pain and between stress and analgesia are enigmatic. This dilemma is compounded in a chronic pain situation where psychological disorders may also be present (47). Pain perception in a given situation to a given stimulus is variable. In addition the strategies used to deal with pain perception are also variable. Some strategies such as imagery or distraction are functional and their use decreases the perception of pain this should lead to less distress but this reduction in stress then decrease the potential for stress induced analgesia. Other strategies such as catastrophysing are dysfunctional and even enhance the perception of pain. Theoretically this should provoke stress induced analgesia but this does not seem to occur as pain perception is increased. Thus the role of stress induced analgesia and pain perception is replete with unanswered questions.

At the present time there is no model that satisfactorially explains the relationship between stress pain and the placebo effect. It is clear that pain is a major stress and coping reactions to this stress influence the pain perception and adaptation to pain (32,15,41,58). Whether pain perception or coping strategies directly and significantly influence placebo analgesia is not clear.

The Placebo and Coping

Coping strategies are associated with health and pain attributions and with self-efficacy beliefs. It is generally acknowledged that the impact of disease on an individual as well as response to treatment is related not only to psychopathology but to psychological characteristics in general (17). One psychological concept that is relevant to placebo effects is that of health locus of control (HLOC). The Multidimensional Health Locus of Control (MHLC) (65) is a scale designed to measure the dimensions of HLOC. It consists of three subscales: internal health locus of control (I-LOC) powerful others health locus of control (P-LOC) and chance health locus of control (C-LOC). Patients with a high I-LOC have a greater belief in their own capacity to cope with or reduce pain. Patients with a strong belief in P-LOC mainly rely on health professionals to reduce their pain. Finally patients with a C-LOC believe that their health situation depends on luck or fate (12).

There is an intuitive notion that good treatment outcomes would correlate with a P-LOC and there is investigative support for this (47). However, there is also evidence that supports a strong correlation between I-LOC and good treatment outcome (27). Recent evidence has shown that LOC style is a significant predictor of perceived pain (6). Subjects with an I-LOC tend to perceive less pain. This explains the occurrence of positive treatment results in patients with an I-LOC as low levels of perceived pain intensity are much easier to control through medication or physical therapy modalities.

LOC in relation to placebo reactivity has not been addressed in the literature. But several factors make the relationship worthy of consideration. For instance LOC is based on personal attitudes or philosophies experiences and beliefs. Ethnicity is a strong predictor of LOC which implies that LOC is also based on social experience (6). All of these factors impact on the placebo effect of treatment.

Anecdotal and experimental evidence supports the contention that the more powerful the intervention is perceived to be the greater is its potential for effect. Hence injections have a greater placebo effect than pills (35). Is it possible that the perceived power of a specific treatment or the power of the person administering the treatment will have a greater placebo effect on patients with a P-LOC? Likewise patients with an I-LOC would be less impressed by external factors and their response to treatment would be primarily due to specific treatment effects. The differential perception of healing power of physical therapy treatments has not been tested. Problems in this type of study relate to adequate blinding of the investigator as investigators themselves influence the placebo response.

The beliefs of patients in their treatment influences treatment efficacy. If the philosophical basis of a treatment programme coincides with the patient's philosophies then treatment is more likely to be sucessful. This notion supports the role of expectancies in placebo effects.

Expectancies of both clinicians and patients are known to influence the placebo response (22,8). Patients who expect a great deal of post-operative pain tend to report a great deal of pain (67). However when patients have high expectations of pain relief a relatively inert substance can induce some analgesia (45). In terms of pain management the provision of accurate information regarding the expected intensity of pain seems to be most effective in reducing the perception of pain (67). Presumably this congruence between expectation and experience is reassuring. Congruence between expectation and perception of pain may reduce the anxiety which would otherwise increase pain perception. Also the "honesty" of the clinician may enhance the patient-clinician relationship. Certainly the quality of this relationship is an important factor in health care.

Peat (48) has discussed the quality of the physical therapist-patient relationship as contributing to the efficacy of treatment. However the non-specific aspects of any physical therapy treatments have not been explored in a quantitative manner. Peat (48) argues that physical therapists have the opportunity to develop quality relationships with their patients because of the one-on-one time. Thus the therapist is possibly the most powerful placebo of all and their influence on outcome is immense.

In addition strong beliefs of the physical therapists in treatment efficacy will actually enhance efficacy (22,48). Presumably unconcious and conscious behaviour by the clinician influences the beliefs and perceptions of the patient. This will be especially powerful if the beliefs of the patient in their treatment are also strong. The question is: does it matter if the treatment efficacy is based on the placebo effect?

It may be argued that as long as the patient improves the reason for the improvement doesn't matter. However professional and ethical constraints should overide this rationale. First the health professional should be aware that they are using a placebo. Secondly there needs to be protection against financial exploitation (13). Therapists and manufacturers should be part of the solution to current health care financial crises not part of the problem. It is important to try to determine the extent of the placebo effects of therapy and how they contribute to the specific effects. Placebo effects of treatments should be recognised and utilised but should they be the "whole event"?

The Extent of the Placebo Effect

How great are the placebo effects? Non-specific effects have been considered to contribute a fixed fraction (one third) to any treatment. This is a myth (63). For example experience may change non-responders to responders (61). The complexity of the placebo response surely suggests that a fixed fraction is unreasonable. The fixed effect myth stems from a paper by Beecher (7). Beecher summarised his own studies to obtain an average placebo effect. However there was a great deal of variation between the studies depending on the methodology and outcome measured. A global level

of "percent of patients satisfactorily relieved by a placebo" was used on which to average and determine the placebo response. Use of percentage scores and global scores can be totally misleading in any situation. But even if they were acceptable outcomes the range reported by Beecher was between 15 and 58% the mean effect of 36% is obviously rather meaningless and even misleading. It seems intuitive that this figure will vary greatly depending on the treatment the methodology and the time frame of assessment. The time frame is particularly relevant as it is suggested that placebo effects extinguish with time (45). Specific outcomes are also important. Subjective outcomes may be more easily influenced by the placebo effect than objective outcomes.

A thorough determination of the extent of the placebo effect in rehabilitation has not been made. The placebo effect has been assessed in a limited and indirect sense through the use of randomised controlled trials. However usually little consideration is given to changes occurring in the control group. It is these changes that give an indication of the placebo effect. Differences between the control and experimental groups are reported but usually only in relation to the positive effects of treatment. The placebo effects of the intervention are not discussed.

The specific effects of a treatment (T), compared to a baseline (O) can be determined by the calculation T-O (34). This calculation represents both the specific and placebo (P) effects of a treatment. Specific treatment effects are represented by the equation T-P. P-O indicates how effective the placebo effect is. It also gives an indication of whether the placebo group has acted as a "true control". Despite its simplicity this model has not been utilised in the clinical literature.

The issue of the adequacy of <u>true</u> control groups in clinical trials is important. Very few studies have utilised untreated groups for comparison (18). Therefore the natural course of the condition can not be determined. In addition a feeling of wellbeing can be evoked simply by being attentive to the patient. An untreated control group must be utilised whenever ethically and practically possible if the findings are to have scientific credibility.

The sensory characteristics of many physical therapies make blinding difficult. Cross-over and comparison designs have been done (46,14,20). However crossover designs remove the blinding effect for the patient and a comparison of treatments does not permit determination of the placebo effect of either treatment. Neither does it permit blinding of the therapist thus treatment biases of the therapist can confound the results.

Based on the literature it is often difficult to determine the specific effects of many types of physical therapies used to modulate pain. Anecdotal reports of treatment success do not control for the non-specific effects of the particular protocol. They fail to allow for the "salesmanship" of the therapist and usually do not take into consideration the natural course of the symptoms. Reports in the literature sometimes allow a comparison between treatment and placebo groups. But it is rarely possible to determine how the placebo effect

differs from a baseline. In order to illustrate the difficulties posed in attempting to extract the specific and non-specific aspects of treatment the examples of transcutaneous electrical nerve stimulation (TENS) and laser will be used.

TENS Laser and Placebo

TENS is one of the most commonly used modalities for the treatment of pain and has been used in physical therapy for more than 20 years. There is a large body of literature on TENS and some conflicting reports on the efficacy of TENS for pain relief. The use of low power laser for analgesia and wound healing has been practised by physical therapists for the last 5-10 years. The specific properties of laser have been used in science and technology for years but the effect of laser on organic tissue is controversial. The analgesic effects of laser are controversial. Many studies have reported no analgesic effect from laser (9,26,25,31). Others have reported an analgesic effect but one that is no greater than that of a placebo or control group (59,37). Finally some studies have reported a definitive analgesic effect from laser (62,38). These conflicting results are generally reflective of a host of methodological differences. Perhaps the most discriminating difference is that of experimental control and outcome measures. The method used to determine success will influence results of the treatment (29).

This problem is compounded in pain measurement. The simplicity of many psychophysical measures of pain intensity or pain relief belies their complexity (68). This is especially true when percentage changes are reported. Percent change biases any measure in favour of lower pain baseline scores. For example, a common criterion for success is given as the achievment of 50% pain relief. On a ten point scale, this success criterion would be met in different ways according to the baseline measure. A pre-test score of two would need to decrease by one point in order to meet the 50% criterion. On the other hand a pre-test measure of eight would need to decrease by four points in order to meet the same 50% success criteria. This represents a clear bias in favour of mild pain. A further problem with these pain scales is the assumption that they are linear scales, which is not true.

Success of treatment effects has also been based on many other criteria such as a decrease in analgesic consumption (11,54) changes in pain threshold and tolerance (53) grip strength (39,24,5) range of motion (42) skin resistance (56) nerve conduction (23) electrophysiologic recording (31) somatosensory evoked potentials (3) and in respiratory function tests (55).

Grip strength has been used in an attempt to obtain an objective test of change in function due to TENS and laser. There is an inherent assumption that the magnitude of grip strength is directly related to pain perception. However, grip strength is also affected by such factors as; the general level of activity the condition of the muscle, joint stiffness and gender.

TENS

Abelson et al (1) measured resting pain pain during gripping and grip strength in patients with rheumatoid arthritis (RA). They conducted a randomised double blind non-crossover study of active and placebo TENS but no other control. Pain at rest and whilst gripping and grip strength was measured pre- and post-test. The authors reported that the TENS group showed significant improvements in pain and grip strength measurements compared to the placebo group. Grip strength measurements improved during testing but returned to baseline values between tests. This probably confirms that the magnitude of grip strength was pain related in these subjects. The authors report a placebo effect of 17% but it is not clear how they arrived at this figure. They also state that both groups had similar baseline measures yet report mean values for baseline pain of 75 mm in the placebo group compared to 60.5 mm in the experimental group. The magnitude of this difference may have biased the efficacy of TENS in favour of the experimental group.

The study by Kumar and Redford (39) was designed to make an objective assessment of pain relief and to evaluate the placebo effects of TENS. The time that a specific weight could be held was measured and provided a baseline measurement. A three part test was applied using the same weight. Each wrist was tested using an active TENS sham TENS and the opposite wrist was loaded during stimulation. If the loading time doubled or more the pain relief was considered to be 100%. The results show that 70% of the wrists with active TENS achieved 50-100% pain relief. This contrasts to 15% of the sham TENS and 10% of the opposite wrists. If the 10% figure is accepted as a baseline figure this indicates a placebo effect of 5% and a specific effect of 60% measured in this manner.

However, as noted earlier there are problems with the use of percentage changes as a measure of success. In addition it is clear that the study was not blind. Patients would be aware of perceptual differences between active and sham TENS. A non-crossover study design was used by Langley et al (40). Patients with RA were again used as subjects and similar measures of pain and grip strength were compared between three groups. One group was assigned to receive high frequency TENS another received low frequency TENS and a third received placebo TENS. Again percentage scores were used to determine the success rate. Placebo TENS resulted in a 37% effect on resting pain. This compares to a 55% effect for high frequency and a 64% for low frequency TENS.

These results translate to a specific effect of 18% and 26% for high and low frequency TENS respectively. Pain whilst gripping did not show such a differential effect. The placebo effect of 72% success as determined by this method was higher than the 64% of both active TENS groups. In addition there was no difference between groups on grip strength. This suggests that pain and grip strength were not directly related in these subjects. This study shows reasonable placebo effects for pain (37%) but a much larger effect on pain

during gripping (72%). This may be an indication of the lack of relationship between pain and grip strength in this sample or the improved blinding in this study. It may also be due to encouragement of effort by the therapist or a positive attitude towards exercise by the patient (40).

Laser

Basford et al (5) used Helium neon laser in an osteoarthritic sample of patients. They treated the small joints in the hand but found no difference in pain or strength measures with laser compared to a sham laser group. The lack of an untreated control group makes it impossible to determine the magnitude of the placebo effect. But there was certainly no specific treatment effect.

These studies illustrate the problems with attempting to determine the magnitude of the specific and non-specific effects of pain relieving modalities in similar populations. The specific treatment effect of laser was zero. The specific treatment effect of TENS ranged between 18% and 60% depending on the outcome and the study. The placebo effect of laser was 100% whereas the placebo effect of TENS ranged between 5% and 72% depending on the outcome.

Controlled trials of the use of laser for pain relief have not been supportive of its use. Haker and Lundeberg (25) in a double-blind study in patients with epicondalgia reported no specific effect of laser. In another double-blind crossover study patients with chronic oro-facial pain were treated with invisible infrared laser (26). Two probes were used one active and one inactive and only the manufacturer knew which was which. The authors reported that the clinical impression or guess about which probe was active was totally wrong! There was no difference between placebo and control group on any of the pain measures.

Discussion

The use of TENS has more support and interesting results. Cooperman et al (11) compared active and control TENS for postoperative pain. Success was rated as excellent (no analgesia) good (a maximum of three doses of demerol) and poor (more than three doses of demerol). They reported an excellent success rate of 34% in the TENS group vs 12% success in the placebo TENS group. Forty two percent of the TENS group obtained a good result vs 20% of the placebo group. Finally 23% of the TENS group obtained poor success vs 67% of the placebo group. The specific effect of TENS can be calculated at 22% for both excellent and good results. Assuming that a true baseline would be zero the placebo effect of TENS would be determined as 12% for an excellent result and 20% for a good result. Positive effects of TENS were also reported in post-operative laminectomy patients (49). They also used percentage pain relief to measure success. Fifty percent or greater pain relief was the criterion determined for success. Based on this criterion the

authors reported an 87.5% success rate for the TENS treated cervical laminectomy group and 79% for the lumbar laminectomy group. They also report a 50% reduction in narcotic consumption in the TENS group compared to controls. These results appear to strongly support a specific effect of TENS however both the placebo effect and specific effect of TENS are embedded in these figures.

In contrast to these positive effects of TENS Smedley and collegues (55) found no difference between TENS and placebo TENS following inguinal hernia repairs. No significant differences in pain measurements opiate requirements and peak expiratory flow measures were found between the active and sham TENS groups.

The discrepant results between this study and previous studies may be due to differences in measuring pain; i.e., pain rather pain relief was measured. This may be a better measure of the effect of pain given that it requires less mathematical manipulation on the part of the patient. This is especially relevant as the patients may still be under the influence of anasthesia or sedation. However, the use of peak expiratory flow may not be a fair method of determining success. The authors note that this test evokes a very sharp and intense pain. Previous research has reported that TENS is less effective for pain that is sharp in quality compared to dull pain (53). Also in this study patients did not manipulate the controls of their TENS units to allow for optimum perception of parasthesia. This may also have influenced the efficacy of TENS. One other point to consider is that post operative pain is usually moderate to severe so it is possible that the effect of TENS is not likely to be major. It has been shown that personality contributes much more to pain perception and narcotic requirements (80% of the variance) than TENS (20% of the variance) (43). Obviously TENS has no effect on personality.

Deyo et al (14) compared active and sham TENS alone or active or sham TENS combined with exercises in a group of patients with chronic low back pain. The active TENS group obtained 47% improvement in pain related measures compared to 42% pain relief for the sham TENS group. The exercise group obtained a 52% improvement in pain related measures. The authors suggest that TENS is no more effective than a placebo and that exercise is just as effective in improving pain related measures. However the "pain related measures" used in this study were actually exercise related pain. In addition the results are compromised by the use of global scores. The global score appears to be the mean of all measures. There is no evidence in support of the validity of this score as a measure of effect. It appears that all measures were given the same weight towards the score. In addition many of the measures used are activity based which gives a positive bias towards the effect of exercise. The use of this measure may also be problematic in measuring the effect of TENS. However this study certainly suggests that the only effect of TENS is the non-specific or placebo effect.

Klein and Eek (37) used laser in a back pain population and reported a similar result. In a double blind controlled trial they found that neither laser nor placebo laser offered any more benefit to patients with chronic low back pain than exercise alone. Specific treatment results of modalities do not appear to be efficacious in low back pain patients. However, the specific effect of both laser and TENS is pain relief. A secondary benefit would be change in function but only if this function is directly related to the pain. Moreover, testing should be done during rather than after the stimulation period in order to determine effect. This is not usually done.

It is clear that a determination of either the specific or the non-specific effects of TENS and laser is a challenge. The magnitude of treatment and placebo effects vary greatly according to the experiemntal design and the criteria used to determine success. Pain relief measures tend to give a much greater measure of success than pain intensity measures. Pain during some measured activity is variable in its response to TENS, whereas laser has no specific clinical effect.

Clinical studies are accepted as having less experimental control than basic science studies. It is interesting to consider the results of experimental studies relating to the purported analgesic effects of TENS and laser. Electrophysiological recordings from nociceptive afferents are not influenced by laser (31) whereas TENS influences afferent transmission in A-beta and A-delta fibres (30). Nerve conduction velocities are not influenced by laser (23) but TENS increases the threshold of the nociceptive flexion reflex (10,21,17). Laser has not influenced experimental pain threshold (9) but TENS has (53) and has not (33) increased experimental pain thresholds. TENS has also been shown to increase pain threshold in laboratory animals (34) and influence somatosensory evoked potentials a robust physiological measure which is thought to correlate well with pain stimulus intensity and self report (3). Experimental studies tend to support a specific effect of TENS based on electrophysiological measures but that support is not present for laser.

In summary laser has no specific effect on the physiological mechanisms subserving pain. However TENS evokes specific physiological effects that can be measured. This supports the contention that laser acts through placebo mechanisms only. Does it matter? Perhaps not as long as there is no harm and there is no financial exploitation. But who wins when patients are treated with laser? Are patients being set up for failure? Or is laser an acceptable form of placebo therapy? Who really wins when a $10 000 laser is purchased (13)? The only clear winners are the electromedical manufacturers and the sales companies.

REFERENCES

1. Abelson K, Langley GB, Sheppeard H, Vlieg M, Wigley RD. Transcutaneous electrical nerve stimulation in rheumatoid arthritis. New Zealand Medical Journal 1983:96;156-158.

2. Al Absi M, Rokke PD. Can anxiety help us tolerate pain? Pain 1991:46;43-51.

3. Ashton B, Piper MC, Warren S, Stewin L, Byrne P. Influence of medical history on assessment of at-risk infants. Developmental Medicine and Child Neurology 1991:33;12-418.

4. Ashton H, Golding JF, Marsh VR, Thompson JW. Effects of transcutaneous electrical nerve stimulation and aspirin on late somatosensory evoked potentials in normal subjects. Pain 1984:18; 377-386.

5. Basford JR, Sheffield CG, Mair SD, Ilstrup DM. Low energy helium neon laser treatment of thumb osteoarthritis. Archives Phys Med Rehabil 1987:68;794-797.

6. Bates MS, Edwards WT, Anderson KO. Ethnocultural influences on variation in chronic pain perception. Pain 1993:52;101-112.

7. Beecher HK. The powerful placebo. Journal of the American Medical Association 1955:159;17;1602-1606.

8. Bootzin RR. The role of expectancy in behaviour change. In: White L, Tursky B, Schwartz GE. (eds) Placebo. Theory research and mechanisms. New York: Guilford Press 1985:196-210.

9. Brockhaus A, Elger CE. Hypalgesic efficacy of acupuncture on experimental pain in man. Comparison of laser acupuncture and needle acupuncture. Pain 1990:43;181-185.

10. Chan CWY, Tsang H. Inhibition of the human flexion reflex by low intensity high frequency transcutaneous electrical nerve stimulation (TENS) has a gradual onset and offset. Pain 1987:28; 239-253.

11. Cooperman AM, Hall B, Mikalacki K, Hardy R, Sader E. Use of transcutaneous electrical stimulation in the control of postoperative pain. The American Journal of Surgery 1977:133;2; 185-187.

12. Crisson JE, Keefe FJ. The relationship of locus of control in pain coping strategies and psychological distress in chronic pain patients. Pain 1988:35;147-154.

13. Devor M. What's in a laser beam for pain therapy? Pain 1990:43; 139.

14. Deyo RA, Walsh NE, Martin DC, Schoenfeld LS, Ramamurthy S. A controlled trial of transcutaneous electrical nerve stimulation (TENS) and exercise for chronic low back pain. The New England Journal of Medicine 1990:322;23;1627-1634.

15. Edwards LC, Pearce SA, Turner-Stokes L , Jones A. The pain beliefs questionnaire: an investigation of beliefs in the causes and consequences of pain. Pain 1992:51;267-272.

16. Evans FJ. The placebo response in pain reduction. In: Bonica J J.(ed) Advances in Neurology Vol. 4 New York: Raven Press 1974:1-42.

17. Facchinetti F, Sandrini G, Petraglia F, Alfonsi E, Nappi G, Genazzani AR. Concomitant increase in nociceptive flexion reflex threshold and plasma opioids following transcutaneous nerve stimulation. Pain 1984:19;295-303.

18. Fahrer H. Analgesic low frequency electrotherapy. In: P. Schlapbach P, Gerber NJ. (eds) Physiotherapy: Controlled Trials and Facts. Rheumatology vol. 14. Basel Karger 1991:150-162.

19. Farrer GR. Psychoanalytic theory of placebo. Disorders of the Nervous System 1965:35;655-662.

20. Fox EJ, Melzack R. Transcutaneous electrical stimulation and acupuncture: Comparison of treatment for low-back pain. Pain 1976:2;141-148.

21. Francini F, Maresca M, Procacci P, Zoppi M. The effects of non-painful ranscutaneous electrical nerve stimulation on cutaneous pain threshold and muscular reflexes in normal men and in subjects with chronic pain. Pain 1981:11;49-63.

22. Gracely RH, Dubner R, Deeter WR, Wolskee PJ. Clinicians' expectations influence placebo analgesia. The Lancet 1985:43.

23. Greathouse DG, Currier DP, Gilmore RL. Effects of clinical infrared laser on superficial radial nerve conduction. Physical Therapy 1985:65;8;1184-1187.

24. Griffin JW, McClure M. Adverse responses to transcutaneous electrical nerve stimulation in a patient with rheumatoid arthritis. Physical Therapy 1981:61;3;354-355.

25. Haker E, Lundeberg T. Laser treatment applied to acupuncture points in lateral humeral epicondalgia. A double blind study. Pain 1990:43;243-247.

26. Hansen HJ, Thoroe U. Low power laser biostimulation of chronic orofacial pain. A double blind placebo cross-over study in forty patients. Pain 1990:43;169-179.

27. Harkapaa K, Jarvikoski A, Mellin G, Hurri H, Luoma J. Health locus of control beliefs and psychological distress as predictors for treatment outcome in low-back pain patients: results of a 3-month follow-up of a controlled intervention study. Pain 1991:46;35-41.

28. Hashish I, Feinman C, Harvey W. Reduction of post-operative pain and swelling by ultrasound: a placebo effect. Pain 1988:83;303-311.

29. Howe J, Frymoyer JW. The effects of questionnaire design on the determination of end results in lumbar spinal surgery. Spine 1985:10;804-805.

30. Janko M, Trontelj JV. Transcutaneous electrical nerve stimulation: a microneurographic and perceptual study. Pain 1980:9;219-230.

31. Jarvis D, NacIvor MB, Tanelian DL. Electrophysiologic recording and thermodynamic modeling demonstrate that helium-neon laser irradiation does not affect peripheral A delta or C-fiber nociceptors. Pain 1990:43;235-242.

32. Jensen MP, Turner JA, Romano JM. Chronic pain coping measures: individual vs. composite scores. Pain 1992:51;273-280.

33. Jette DU. Effect of different forms of transcutaneous electrical nerve stimulation on experimental pain. Physical Therapy 1986:66;2;187-190.

34. Jorum E, Shyu B-C. Analgesia by low-frequency nerve stimulation mediated by low-threshold afferents in rats. Pain 1988:32;357-366.

35. Joyce CRB. Non-specific aspects and clinical pharmacology. In: Shepherd M, Sartorious N. (eds) Non-Specific Aspects of Treatment. Toronto: Hans Huber 1989:57-94.

36. Kantor TG, Sunshine A, Laska E , Meisner M, Hopper M. Oral analgesic studies. Clinical Pharmacological Therapeutics 1966:7; 447-454.

37. Klein RG, Eek BC. Low energy laser treatment and exercise for chronic low back pain: double blind controlled trial. Arch Phys Med Rehabil 1990:71;34-37.

38. Kleinkort JA. Laser acupuncture: Its use in physical therapy. American Journal of Acupuncure 1984:12;1;51-55.

39. Kumar VN, Redford JB. Transcutaneous nerve stimulation in rheumatoid arthritis. Archives of Physical Medicine and Rehabilitation 1982:63;595 - 596.

40. Langley GB, Sheppeard H, Johnson M, Wigley RD. The analgesic effects of transcutaneous electrical nerve stimulation and placebo in chronic pain patients. Rheumatology International 1984:4;119-123.

41. Lawson K, Reesor KA, Keefe FJ, Turner JA. Dimensions of pain-related cognitive coping: cross-validation of the factor structure of the Coping Strategy Questionnaire. Pain 1990:43:195-204.

42. Leandri M, Parodi CI, Corrieri N, Rigardi S. Comparison of TENS treatments in hemiplegic shoulder pain. Scandinavian Journal of Rehabilitation Medicine 1990:22;69-72.

43. Lim AT, Edis G, Kranz H, Mendelson G, Selwood T, Scott DF. Postoperative pain control: contribution of psychological factors and transcutaneous electrical stimulation. Pain 1983:17;179-188.

44. Magni G. On the relationship between chronic pain and depression when there is no organic lesion. Pain 1987:31;1-21.

45. Mayer DJ, Price DD. The neurobiology of pain. In: Snyder-Mackler L, Robinson AJ. (eds) Clinical Electrophysiology. Electrotherapy and electrophysiologic testing. Baltimore: Williams and Wilkins 1989:139-201.

46. Melzack R, Jeans E, Stratford JG, Monks RC. Ice massage and transcutaneous electrical stimulation: comparison of treatment for low back pain. Pain 1980:9;209-217.

47. Nagy VT, Wolfe GR. Cognitive predictors of compliance in chronic disease patients. Medical Care 1984:22;912-921.

48. Peat M. Physiotherapy: art or science? Physiotherapy Canada 1981:33;3;170-176.

49. Richardson RR, Siquera EB. Transcutaneous electrical neurostimulation in postlaminectomy pain. Spine 1980:5;4;361-365.

50. Rosenthal R. Designing analyzing interpreting and summarizing placebo studies. In: White L, Tursky B, Schwartz GE. (eds) Placebo. Theory research and mechanisms. New York: Guilford Press 1985:110-136.

51. Sartorius N. Foreword. In: White L, Tursky B, Schwartz G. (eds) Placebo. Theory Research and Mechanisms. New York: Guilford Press 1985:vii - viii.

52. Shepherd M, Sartorius N. Introduction. In: Shepherd M, Sartorius N. (eds) Non-specific Aspects of Treatment. Toronto: Hans Huber 1989:1-4.

53. Simmonds MJ, Wessel J, Scudds RA. Quality of pain and the effectiveness of pain. Physiotherapy Canada 1992:44;3;35-40.

54. Simmonds MJ, Scudds RA. A TENS home loan programme evaluation (Abstract) Physiotherapy Canada 1990:42;(Suppl)9.

55. Smedley F, Taube M, Wastell C. Transcutaneous electrical nerve stimulation for pain relief following inguinal hernia repair: A controlled trial. European Surgical Research 1988:20;233-237.

56. Snyder-Mackler L, Bork C, Bourbon B, Trumbore D. Effect of helium-neon laser on musculoskeletal trigger points. Physical Therapy 1986:66;7;1087-1090.

57. Tierney G, Carmody J, Jamieson D. Stress analgesia: the opioid analgesia of long swims suppresses the non-opiod analgesia induced by short swims in mice. Pain 1991:46;89-95.

58. Turner JA, Clancy S. Strategies for coping with chronic low back pain: relationships to pain and disability. Pain 1986:24;355-364.

59. Vasseljen O. Low-level laser versus traditional physiotherapy in the treatment of tennis elbow. Physiotherapy 1992:78;5;329-334.

60. Voudouris NJ, Peck CL, Coleman G. The role of conditioning and verbal expectancy in the placebo response. Pain 1990:43;121-128.

61. Voudouris NJ, Peck CL, Coleman G. Conditioned response models of placebo phenomena. Pain 1989:38;109-116.

62. Walker J. Relief from chronic pain by low power irradiation. Neuroscience Letters 1983:43;339-344.

63. Wall PD. The placebo effect: an unpopular topic. Pain 1992:51;1-3.

64. Wallace LM. Surgical patients' expectations of pain and discomfort: does accuracy of expectations minimise post surgical pain and distress? Pain 1985:22;363-373.

65. Wallston KA, Wallston BS, De Vellis R. Development of the multidimensional health locus of control (MHLC) scales. Health Education Monogram 1987:6;160-170.

66. Wickramasekera I. A conditioned response model of the placebo effect: Predictions from the model. In: White L, Tursky B, Schwartz G. (eds) Placebo. Theory Research and Mechanisms. New York: Guilford Press 1985:255-287.

67. Widerstrom EG, Aslund PG, Gustafsson LE, Mannheimer C, Carlsson SG, Andersson SA. Relations between experimentally induced tooth pain threshold changes psychometrics and clinical pain relief following TENS. A retrospective study in patients with longlasting pain. Pain 1992:51;281-287.

68. Wilson PR. Pain research - The science and the art. The Clinical Journal of Pain 1990:6;171-172.

CHAPTER EIGHT

SPINAL MOTION TESTING AND TREATMENT[7]

Introduction

Palpation of spinal structures and spinal motion are fundamental to the assessment and treatment of spinal disorders. Manual application of forces to the spine has been practised for hundreds of years but little is known about the forces used the specific effect of these forces on biologic tissue and the accuracy of therapists in assessment of applied force. The sensitivity, reliability, validity and efficacy of many mobilization skills are controversial in part because they have been subjected to little scientific scrutiny. Assessment and treatment based on manual palpation are accepted untested by some clinicians (13,7,1) whilst being totally dismissed by others (21). Although motion sense may be the "culmination of palpatory skills and the limiting factor in the art of maniplulation" (1) it is still based on faith and the testimony of true believers rather than on science (24). Of concern is that few studies have empirically addressed static or motion palpation.

The key purpose of passive motion palpation is to assess the quality of motion between adjacent articular segments (22) in making judgements on tissue compliance and to determine whether pain is evoked by the test. There is a low level of reliability associated with many motion tests (22,12.8,6,19) unless it is a test which relies on patient response as determined by pain and tenderness (12,23). However patient response will vary with the amount of force used during testing. The greater the force used the greater the possibility of detecting motion and evoking pain (20). Patient response will also vary with the location of the applied force and there is inconsistency in locating even key landmarks such as the posterior superior iliac spine (26,25). In addition Simmonds (26) has shown experimentally that the identification of a lumbar spinous process is associated with an error of plus or minus one vertebral level. The reliability of location will improve if the patient participates and complains about pain or tenderness. However the complaint of pain or tenderness will be influenced by the amount of force applied.

Standardization of palpation technique is obviously imperative. Yet despite extensive clinical use many techniques are not well described or recorded (22). Text book descriptions of assessment and treatment techniques are qualitative. e.g. (17,18). Terms such as "gentle distraction", "firm", "slow" or "steady" forces are purely subjective and may vary with the tactile acuity and strength characteristics of the physical therapist. No quantitative physical characteristics of applied forces are documented in these descriptions which

---

[7]A version of this chapter has been submitted for publication. Simmonds Kumar and Lechelt 1993. Physical Therapy

makes standardization of testing procedures difficult. This is compounded by the fact that manual skills are taught by "clinical experts" who superimpose idiosyncracy on the technique.

Finally reliability measures are complicated by the fact that repeated testing of biological tissue may lead to an actual change in musculoskeletal stiffness (10). This is true change but it will be reflected in low levels of reliability.

There is a clear need for controlled systematic study quantitative documentation and standardization of mobilization techniques. Many aspects of manual therapy remain unanswered. Questions relate to the specific techniques and to their method of application. Also unanswered are questions that relate to the feedback systems utilised by the therapist i.e. the sensorimotor perceptual and interpretive skills.

The use of mechanical models provides a useful and well controlled experimental paradigm which can be used to measure the characteristics of the techniques and the perceptual abilities of the therapist. Harvey and Byfield (8) utilised a cadaveric vertebral spine which they modified with screws and bushings to control specific segmental motion. The model was covered with a chamois leather in order to eliminate visual cues. Examiners were required to determine the presence or absence of "fixation" at each lumbar and lumbosacral level. The observed rate of correct motion palpation was fairly high (81%) but so was the rate expected by chance (71%). This study was flawed in several respects. Although specificity was high, sensitivity was low indicating a failure to identify the fixation. This could be due to the stiffness characteristics of the model or the lack of a physiological feel to the motion. Neither stiffness nor magnitude of motion were reported. However the authors did report that the model allowed no shear motion. It is possible that the lack of sensitivity was due to problems with the model rather than limitations in the skills of the therapist. These concerns limit the generalizability of results. Another failure in modelling palpation was Evans's (4) attempt to simulate resistance to motion but his model had problems with nonlinearity and hysteresis thereby making any results suspect.

An important contribution to the objective evaluation of mobilization skills was reported by Matyas and Bach (19). They used an indirect method for estimating applied force during passive intervertebral mobilization by having therapists stand on a force platform thereby allowing for the calculation of the forces applied to the patient. One important finding from this study was confirmation of the large range of intra- and inter-therapist variability. The authors report that the applied force varied between therapists by a factor of nine (19). Lee Moseley and Refshauge (15) used the same method of measurement and provided feedback to therapists using an oscilloscope. The feedback resulted in a greater degree of consistency in the application of a mobilizing force. A similar positive effect of training was also reported by Keating and collegues (11). This suggests that therapists can learn to quantify

applied forces and this will lead to a more consistent approach for evaluating treating and communicating joint behaviour.

Mechanical models provide a useful experimental paradigm to examine the sensory and motor aspects of palpation skills. They provide a controlled standardised test situation allowing precise and accurate measurements of the characteristics of forces used. In addition, they provide a method for measuring the perceptual accuracy of the therapist both in force application and in motion detection.

These factors are important in clinical applications. The amount of applied force will influence the perception of stiffness of a joint. Stiffness is perceived by feeling the resistance to motion. It is the ratio of change in applied force to a change in displacement (14). If stiffness is accepted as an indication for mobilization then it is essential to determine the accuracy of both force application and motion detection. Perceptual accuracy of the therapist can best be determined in vitro because of the amount of control that can be exerted over the test situation. Once the characteristics of a specific technique are determined they can be taught in a standardised manner and then used and tested for specific effect and efficacy in vivo.

The purpose of this study is to determine: 1. the magnitude of the forces used by physical therapists during spinal motion testing and treatment under different conditions of stiffness; 2. the resultant vertebral motion; 3. the accuracy of perception of applied force and motion detection; 4. the simulatory quality of physiological motion in the mechanical spinal model.

METHOD

Experimental design

The experiment was a 3x5x3 repeated measures randomised factorial design. There were three levels of stiffness five grades of mobilizations and three replications.

Subjects

Three male and seven female physical therapists participated in this experiment; all were familiar with the Maitland mobilization techniques used in the experiment. Following recruitment they were apprised of the research questions and familiarised with the spinal model. An informed consent was signed prior to participation. They were familiarised with the spinal model for a few minutes prior to the experiment.

Tasks

Therapists applied three test grades of motion to determine the range available and the resistance to motion. Grades of mobilization as described by Maitland (16) were then applied in a randomised order. The descriptions provided to the therapists were written as follows: Grade 1 is a small-amplitude

movement near the starting position of the range. Grade 2 is a large-amplitude movement which carries well into the range. It can occupy any part of the range that is free of any stiffness or muscle spasm. Grade 3 is also a large-amplitude movement but one that does move into stiffness or muscle spasm. Finally grade 4 is a small-amplitude movement stretching into stiffness or muscle spasm (18). Grade 5 was the initial test grade.

The mobilization force was applied through both thumbs or through the pisiform bone of the wrist, whichever was the customary technique used by the therapist.

Equipment

The mechanical spinal model was specially designed and fabricated for this experiment (Figure 8.1). In essence a vertebra was mounted on top of a specially designed spring resisted housing. This was covered by a moulded plastic back brace with a three cm wide and twelve cm long hole in the brace at the level equivalent to the lumbar spine. This hole was covered by high density rubber which allowed the therapist to palpate the spinous process of the vertebra which was underneath (Figure 8.2).

The main structure of the model was a round brass block 12.5 cm in diameter and 12.5 cm in length. At one end of the block a 7.7 cm diameter bored hole was machined 6.5 cm deep in the middle of the block. At the base of this hole six smaller precise holes were machined in order to accomodate up to six springs. Each hole was 3 cm deep and 1.5 cm in diameter. A variable number of springs were placed in the model in a balanced manner in order to simulate different levels of stiffness. All springs were identical. They each had a spring constant of 222 Newtons /cm were 8 cm in length and had an external diameter of 1.5 cm.

A precisely machined aluminium block 7.8 in diameter and 6.5 cm in length was fitted inside the main brass hole. At the base of this aluminium block machined impressions for the springs which corresponded to those in the brass base were made. At the top of the aluminium block a rounded rectangular hole was machined. A specially constructed load cell was fitted into this hole. Two guide bars protuded from this aluminium block in an upward direction. A second aluminium block of the same diameter but 2.5 cm in length was placed above the load cell. Two holes in the side fitted the guide bars from the lower block. The guide bars ensured a vertical motion. At the bottom of this aluminium block was a small circular metal protusion which made contact with the load cell. At the top of the uppermost aluminium block a round hole was machined out and a resin mounted vertebra was firmly fitted.

A linear variable differential transducer (LVDT) was mounted on the side of the brass block in order to measure displacement of the vertebra. This was coupled through a thin aluminium bar to the movable aluminium insert block. Finally this device was placed on a wooden platform and was covered by the polypropylene shell moulded into the shape of a human trunk.

The load cell and LVDT were sampled at a frequency of 50 Hz through a Data Translation 2801 A/D data acquisition board and fed into an IBM computer.

Procedure

Calibration of the LVDT and the load cell was conducted prior to testing each subject. The spinal model was positioned on an adjustable plinth allowing the therapist to assume their normal working posture. For each of the three levels of resistance the therapist first tested the allowable range of motion available at the motion segment. This was termed the maximum test and was done three times. Other grades of mobilization were applied in a randomised order. One, two or three springs were placed in the model. Therapists were instructed to use the same amount of force that they would normally use during clinical spinal motion testing. All treatment grades (1-4) were applied for ten seconds. At the end of each set of three repetitions the therapists were asked to estimate the amount of peak force that they had applied for the mobilization and also estimate the magnitude of displacement produced.

The degree to which the mechanical spinal model simulated normal motion was assessed using two 11 point numerical rating scales. For each level of stiffness, PTs were asked to grade the spinal model on according to how well the model simulated normal physiologic motion. Zero was labelled "not at all' and 10 was labelled "perfect simulation". In order to measure perceived joint stiffness therapist were asked to grade the model on a 0-10 scale. The zero point was labelled "ankylosed" and 10 was labelled "totally unstable".

Analysis

Descriptive statistics were computed for the applied forces and resultant displacement for each grade of mobilization and for each stiffness condition. Data on the perception of applied force and resultant displacement were computed in the same manner.

A repeated measures univariate ANOVA (3 levels of stiffness, 5 levels of mobilisation grades, and 3 repetitions) was computed on the quantitative data with post-hoc least significant difference tests calculated where indicated. In addition, paired t-tests were performed to test for differences between the perception of force and motion and the measured force and displacement.

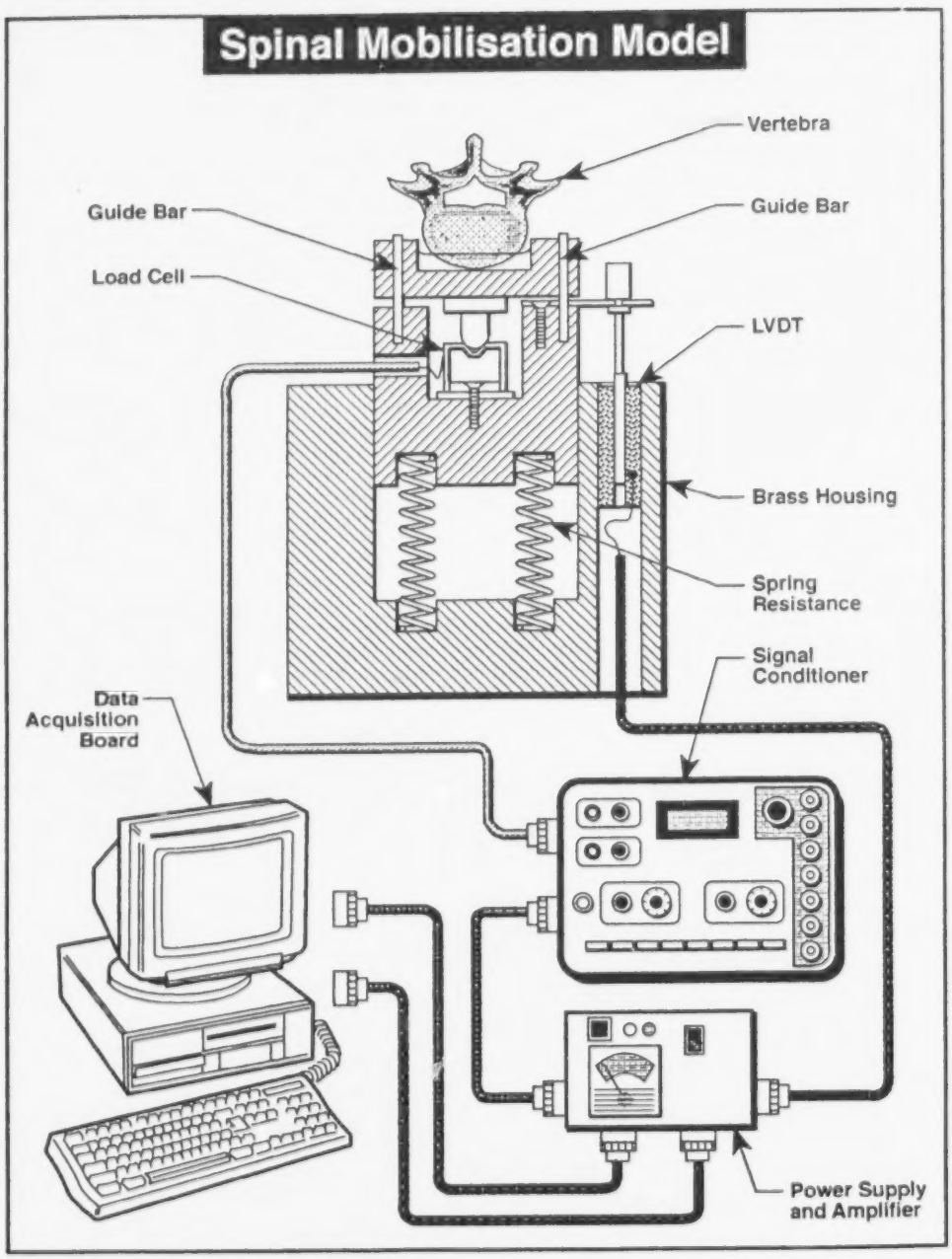


Figure 8.1 Schematic of spinal mobilization model.

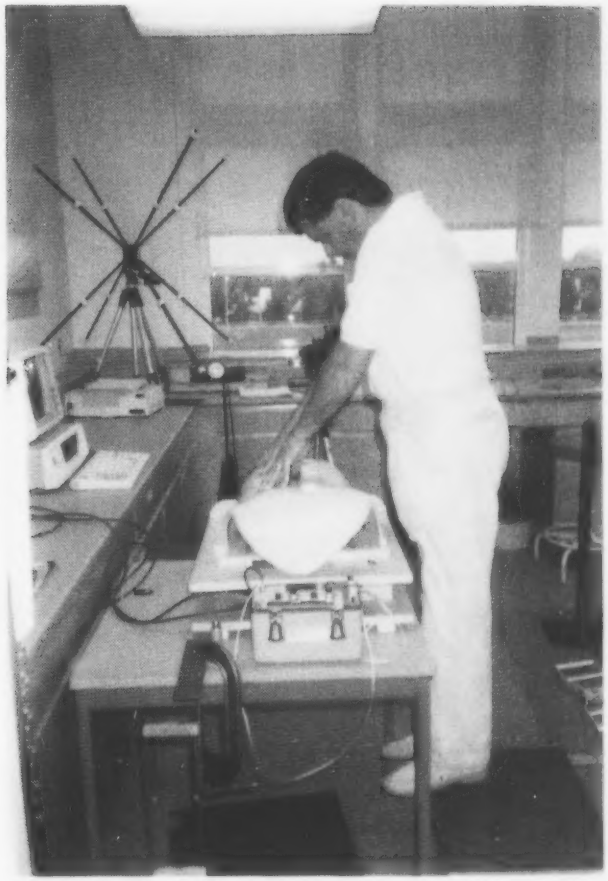


Figure 8.2 Photograph of therapist applying the mobilization technique to the model

RESULTS

The descriptive statistics for peak force and displacement for each grade of mobilization and for each level of stiffness are presented in Tables 8.1 and 8.2 respectively. In general the mean peak force values are lowest in the least stiff condition across all grades of mobilization (57.6 - 120 Newtons). The force values are most similar in the other two stiffness conditions 82 - 178 Newtons and 81 - 161 Newtons respectively.

The applied force generally increased in a stepwise fashion for each grade of mobilization (see Table 8.1). The greatest force values occurred in the maximum test. An exception occurred in the stiffest condition in which the highest force was applied for grade 4 mobilization (161.98 Newtons).

The amount of displacement was greatest in the least stiff level (see Table 8.2). The magnitude ranged from 2.25 - 3.82 mm. for grade 1 and the maximum test respectively. The magnitude of displacement was less for the other two stiffness levels. Moreover the displacement increased with the increase in grades of mobilization.

The anova showed statistically significant effects for mobilization grade and stiffness level but no significant interactions. Post-hoc least significant difference tests were carried out and revealed significant differences between stiffness levels 1 and 2 and between 1 and 3 but not between stiffness levels 2 and 3. This pertained both to the force application and the resulting displacement. There were significant differences between grade 1 and all other grades of mobilization. Grade 2 was significantly different from grades 3, 4 and maximum.

Paired t-tests were carried out between the perceived and actual force for each grade of mobilization. All were significantly different ($p < .0001$) (see Figure 8.3). Perceived and actual levels of displacement were tested in the same manner and were all significantly different ($p < .005$) except for grade 1 ($p = .238$) (see Figure 8.4).

Finally the similarity between human spinal motion and that of the model was assessed by the therapists. Out of a perfect 10, the mean for stiffness levels 1 2 and 3 was 6.45, 4.67, and 2.63, respectively (see Table 8.3). The amount of perceived joint stiffness (between 0 = ankylosed and 10 = totally unstable) was judged to be 5.42, 4.88, and 3.25 respectively for the least to the most stiff level (see Table 8.3).

Table 8.1: Descriptive statistics (Newtons) of peak applied force for each for each grade of mobilization and each level of stiffness (n=10).

Stiffness level	Grade 1	Grade 2	Grade 3	Grade 4	Max. test
Least stiff					
Mean	57.59	85.75	116.16	108.24	120.12
S.D.	40.45	54.15	76.95	64.69	82.07
Minimum	1.74	3.92	10.95	15.80	24.45
Maximum	131.32	209.37	302.20	258.72	360.54
Medium stiff					
Mean	82.43	106.44	155.59	141.92	178.27
S.D.	45.79	53.61	95.10	70.75	87.92
Minimum	7.03	26.74	31.82	45.21	46.73
Maximum	171.10	224.68	371.75	303.85	360.54
Most stiff					
Mean	81.27	112.09	144.56	161.98	154.99
S.D.	57.76	53.72	88.44	79.68	86.32
Minimum	.53	23.01	10.95	30.75	46.06
Maximum	203.41	231.85	357.42	292.41	360.54

Table 8.2: Descriptive statistics (mm) of peak displacement for each for each grade of mobilization and each level of stiffness (n=10).

Stiffness level	Grade 1	Grade 2	Grade 3	Grade 4	Max. test
Least stiff					
Mean	2.25	2.81	3.57	3.45	3.82
S.D.	1.16	1.46	1.88	1.86	1.61
Minimum	.15	.21	.07	.56	.04
Maximum	4.34	5.63	7.85	8.99	6.31
Medium stiff					
Mean	1.85	1.79	2.11	2.01	2.52
S.D.	1.62	1.26	1.15	1.24	1.69
Minimum	.06	.42	.28	.43	.17
Maximum	5.14	5.24	4.56	6.04	8.31
Most stiff					
Mean	1.95	1.96	1.88	2.20	2.10
S.D.	1.43	1.41	1.29	1.87	.96
Minimum	.09	.37	.21	.27	.36
Maximum	4.45	6.00	4.20	9.04	4.04

Table 8.3. Means of therapists assessment of spinal model for simulation of physiological motion (0=not at all 10=perfect) and vertebral stiffness (0=ankylosed 10=totally unstable).

Stiffness level	Physiologic Motion	Stiffness
Least stiff	6.45	5.42
Medium stiff	4.67	4.88
Most stiff	2.63	3.25

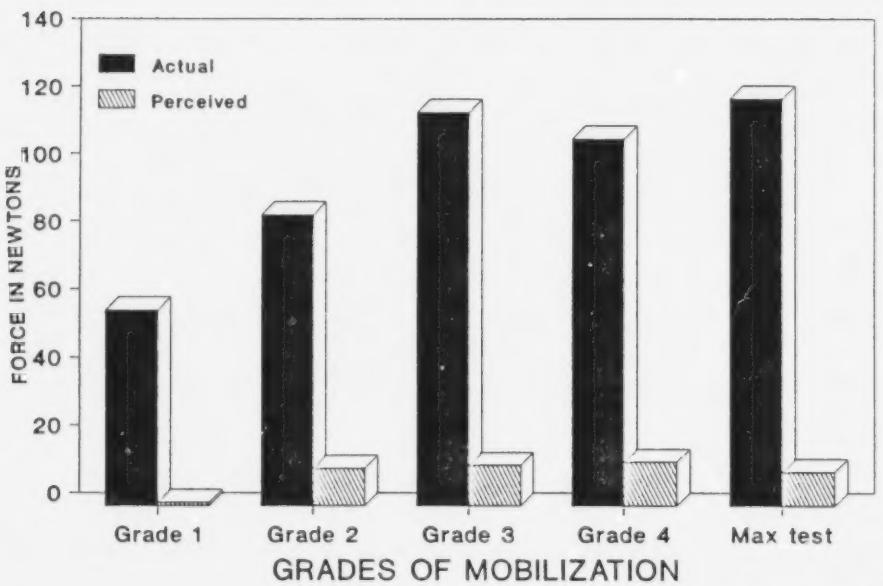
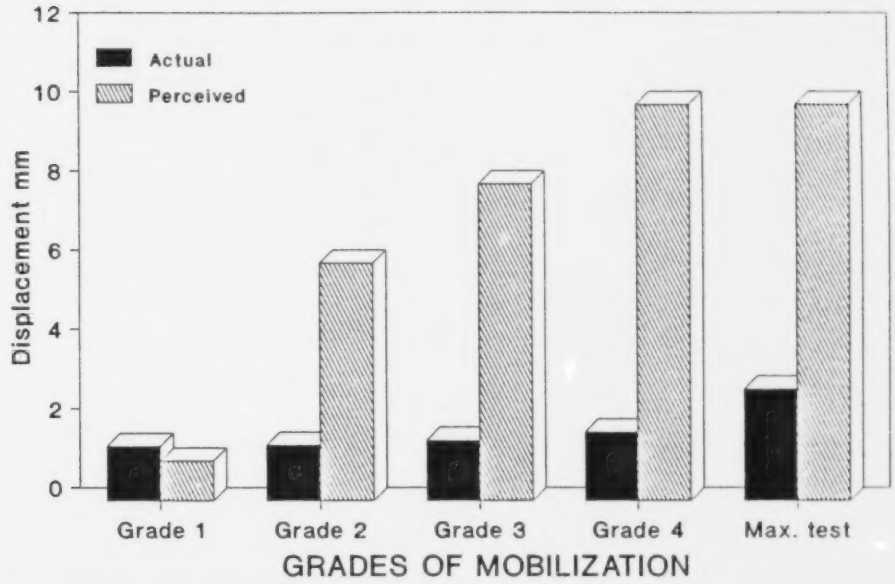


Figure 8.3 Illustrates the difference between the perception of applied force and the actual applied force (Newtons) for each grade of mobilization



Averaged across stiffness levels

Figure 8.4 Illustrates the difference between the perception of displacement and the actual displacement (mm) for each grade of mobilization

DISCUSSION

Force

The mean peak force values measured across grades and stiffness levels ranged between 57.59 to 178.27 Newtons. However, there was a substantial range of inter-therapist variability. For example, for grade 1 mobilization therapists varied between 1.74 and 131.32 Newtons while at grade 4 the range was 15.80 - 258.72 Newtons. There are few published reports that have quantified the forces used during mobilizations so comparison between studies is limited. Lee et al (15) reported an "ideal" grade 2 mobilization of L3 was 33.3 Newtons and Matyas and Bach (19) reported peak forces of 200 Newtons. Threlkeld (27) reported mean forces of 158.8 and 107.7 Newtons for grade 1 and 417.5 and 267.5 Newtons for grade 4 mobilizations. These forces were applied by two manual therapists on the thoracic spine. The forces reported by Threlkeld (who used 2 subjects) are much higher than those utilised by Lee (who used one subject). However, this can be explained by the extent of inter-therapist variability which is revealed in the present study.

It is important to note that the variability in force reported in this study occurred even under highly standardised and controlled conditions. Despite the variability there were significant differences in the applied forces between specific grades of mobilization. Grade 1 is significantly different from all other grades and grade 2 is different from grades 3, 4 and maximum. There was no difference between peak values of grades 3, 4 and maximum. Theoretically these latter grades should have similar peak values given they are applied to the end of the available range.

The results indicated that the Maitland concept of the difference between grades was being applied fairly uniformly. In addition, the differences in magnitude were a reflection of individual interpretation due to the lack of quantitative information.

These sizable differences in magnitude could be remedied if the characteristics of the forces were quantified and taught in a standardised manner. Quantified training with feedback has been shown to improve the consistency and accuracy of applied forces (15,11) and this skill can be retained (15).

The difference in the magnitude of force application between grades was also influenced by the degree of stiffness. There was a significant difference between the least stiff level and the other two stiffness levels. This may suggest that there was a limit to the amount of force that a therapist applied in order to avoid tissue damage.

Given that a force is applied to the spine the question remains as to what is the effect of that force. The explanations have changed radically in recent years (28). There is now less emphasis on unproven dictums such as the restoration of joint alignment and the reduction of nuclear protusions (28). More recent rationale for the use of manual therapy is based on the fact that

biological tissue thrives on stress and motion (27,28). Manual therapy provides both. Unfortunately there is no clinical evidence that shows manual therapy actually achieves this. A related and important issue in regards to magnitude of force is Threlkelds (27) report that connective tissue would begin to experience microfailure between 224 to 1,136 Newtons. It should be noted that the mean values of applied forces in the present study were below the minimum level albeit individual therapists entered this microfailure range. Several points should be considered in regards to this issue. First, the range of microfailure is very large. Moreover, it is not clear whether those which are based on isolated tissue samples and mathematical modelling are accurate in vivo. Further, as these values are based on normal connective tissue it is reasonable to suggest that patients who attend physical therapy will have some compromise to their connective tissue which may alter the biomechanical properties.

Therefore it is possible that these values will have little relationship to the microfailure values occurring in patients with back trouble. Given that the biomechanical properties of connective tissue are altered in pain and injury states it is known that the best way to strengthen these tissues is to stress them. The problem remains of determining the amount of force that is beneficial and not detrimental. This needs to be done under a variety of different pathological conditions and across patient somatotypes (27).

The extent of variability between therapists in their applied force does not support an argument for a very specific effect for Maitland type grades of mobilization. It may account in part for the equivocal results of manual therapy in the literature. Interestingly in a review of the efficacy of manual therapy Di Fabio (3) profiled the patient who would most likely benefit from manual therapy. He stated that patients with low back pain of less than one month duration, central or paravertebral pain distribution, no previous exposure to spinal manipulation and no pending litigation or workers compensation would be most likely to benefit from manual therapy (3). This is precisely the profile of the patient in whom spontaneous resolution of symptoms is likely to occur.

Displacement

The extent of vertebral motion obviously varied with the amount of applied force and stiffness. There was a general and expected trend for motion to decrease as stiffness increased. The mean extent of anteroposterior (AP) vertebral motion (1.79 - 3.82 mm.) was consistent with AP translation occurring in normal individuals (2,29,9,5) However, as with force there was substantial variability in the data.

The displacement values were much less than those reported by Lee and Evans (4) using a spinal mobiliser with LVDT instrumentation. They reported displacement values between 11.06 and 12.61 mm and comment "the data obtained are a good representation of the true intervertebral displacements" (14). The values reported by Lee and Evans are likely movements of extension which is occurring at multiple levels rather than

segmental displacement in that although skin and soft tissue compression is included in these values it is unlikely to be significant over a spinous process.

It was noted earlier that the applied force was consistent with the Maitland concept of mobilization grades. The same trend across grades of mobilization is seen in the least stiff condition but not in the other two stiffness conditions. This may be because the stiffer conditions were too stiff and did not reflect in vivo motion as well. The latter were not rated as highly by therapists in terms of their similarity to normal vertebral motion, 4.67 and 2.63 compared to 6.47 for the least stiff level.

Perception of force and motion

Quantitification of the applied forces and the resultant motion that occurs under specific conditions determines the biological consequences of therapist action during mobilizations. It also allows for further biomechanical testing which can advance the science of manual therapy. However, it is also necessary to establish the relationships between the perceptions therapists have of their interventions and the actual quantification of the intervention and the subsequent motion.

As a group therapists consistently and significantly underestimated the amount of force that they were applying. This discrepancy was present across all grades of mobilization and across all stiffness conditions. There were also a consistent and significant overestimation of the amount of motion that was perceived compared to that which was measured. The sole condition in which there was no difference was for grade 1 when little or no motion was expected. The general level of discrepancy likely reflects the lack of objective feedback in learning and using the technique.

All therapists expressed difficulty in estimating the amount of force that they applied. Many stated that they thought only in terms of grades of mobilizations and this was indicated in the data. However, it was also evident from the data that therapists did not agree on the magnitude of these specific grades.

All but one therapist underestimated the amount of applied force. It is not clear why this therapist overestimated the force. However, this therapist made frequent reference to how she was using her body weight during the mobilization. She then appeared to use this as her baseline reference point. This was interesting as she was also one of the smallest therapists that participated in the experiment.

Motion perception was generally overestimated. The exception was for a grade 1 mobilization. It is possible that this underestimation was not based on sensory cues but rather based on the knowledge that joint motion was not the aim of grade 1 mobilizations.

Therapists expressed less difficulty in estimating the amount of motion compared to force estimation. However, there was significant error associated with the estimation. On an individual basis the amount of motion was

underestimated by six therapists and overestimated by 4. However, these are mean values which obscure potential individual trends across grades of mobilization or across stiffness conditions. The magnitude of discrepancy seems greater in those that overestimated the amount of displacement. It is possible that these subjects consistently overestimated perceived motion. Clearly there is a need for psychophysical study in relation to mobilization. This will allow for a determination of the sensory acuity of therapists and to determine how they make clinically relevant judgements.

CONCLUSIONS

The magnitude of applied force was found to vary with the grade of mobilization and with resistance. However there was a large range of variability between therapists. The perception of applied force and resultant motion was inaccurate. There was a consistent bias in underestimating the amount of applied force and overestimating the amount of motion.

The use of a spinal model provided a useful method for characterising the forces and displacements used during mobilisation. Once the physical characteristics of the forces used have been quantified accurate testing of the effects of these forces on different tissues could be determined. In this way a more specific evaluation of the efficacy of manual therapy can be carried out.

The use of such a model also provided an excellent teaching tool which can be used to train therapists in a standardised manner. This can be done under many different conditions of stiffness. Standardised and reliable application of mobilization techniques is a prerequisite for the controlled clinical testing of manual therapy. Finally the spinal model can be used in the psychophysical testing of the sensory motor and perceptual skills used in physical therapy.

REFERENCES

1. Beal MC. Motion sense. J. Amer Osteo Assoc. 1953:53;151-153.

2. Boden SD, Wiesl SW. Lumbosacral segmental motion in normal individuals. Have we been measuring instability properly? Spine. 1990:15;571-576.

3. DiFabio RP. Efficacy of manual therapy. Phys Ther. 1992:72;853-864.

4. Evans DH. The reliability of assessment parameters: accuracy and palpation technique. In Grieve G. ed. Modern Manual Therapy. Edinburgh; Churchill Livingstone; 1986:498-502.

5. Frymoyer JW, Pope MH, Wilder DG. Segmental instability. In: Weinstein JN, Wisel SW. (eds.) The Lumbar Spine. Philadelphia; WB Saunders; 1990:612-636.

6. Gonella C, Paris SV, Kutner M. Reliability in evaluating passive intervertebral motion. Phys Ther.1982:62;436-444.

7. Grieve GP. Common Vertebral Joint Problems Edinburgh: Churchill Livingstone 1981.

8. Harvey D, Byfield D. Preliminary studies with a mechanical model for the evaluation of spinal motion palpation. Clin Biomech. 1991:6:79-82.

9. Hayes MA, Howard TC, Gruel CR, Kopta JA. Roentgenographic evaluation of lumbar spine flexion-extension asymptomatic individuals. Spine. 1989:14;327-331.

10. Johnston WL. The role of static and motion palpation in structural diagnosis. In: Workshop on the Research Status of Spinal Manipulative Therapy. Bethesda; Maryland; 1975:249-253.

11. Keating J, Matyas TA, Bach TM. The effect of training on physical therapists' ability to apply specified forces of palpation. Phys Ther. 1993:73;38-46.

12. Keating JC, Bergmann TF, Jacobs GE, Finer BA, Larson K. Interexaminer reliability of eight evaluative dimensions of lumbar segmental abnormality J Manip Physiol Ther. 1990:13;463-470.

13. Lee D. The Pelvic Girdle. Edinburgh: Churchill Livingstone; 1989:15-91.

14. Lee R, Evans J. Load displacement-time characteristics of the spine under posteroanterior mobilization. Aust J Physiother. 1992:38;115-123.

15. Lee M, Moseley A, Refshauge K. Effect of feedback on learning a vertebral joint mobilization skill. Phys Ther. 1990:70;97-104.

16. Lee M, Evans J. Load-displacement-time characteristics of the spine under posteroanterior mobilization. Aust J Physiother. 1992:38;115-123.

17. Magee DM. Orthopaedic Physical Assessment (2nd edition) Philadelphia; W.B.Saunders; 1992.

18. Maitland GD. Vertebral Manipulation. 5th Ed London; Butterworths; 1986.

19. Matyas TA, Bach TM. The reliability of selected techniques in clinical arthrometrics. Aust J Physiother. 1985:31;175-200.

20. McClure PW, Rothstein JM, Riddle DL. Intertester reliability of clinical judgements of medial knee ligament integrity. Phys Ther. 1989:69;:268-275.

21. McKenzie RA. Mechanical diagnosis and therapy for low back pain: toward a better understanding. In:Twomey LT, Taylor JR, eds. Physical Therapy of the Low Back. New York Churchill Livingstone1987:157-173.

22. Mootz RD, Keating JC, Kontz HP, Milus TB, Jacobs GE. Intra-and interobserver reliability of passive motion palpation of the spine. J Manip Physiol Ther. 1989:12;440-445.

23. Potter NA, Rothstein JM. Intertester reliability for selected clinical tests of the sacroiliac joint. Phys Ther. 1985:65;1671-1675.

24. Rothstein JM. Manual therapy: A special issue and a special topic. Phys Ther. 1992:72:839-841.

25. Simmonds MJ, Kumar S. Location of body structure by palpation. A reliability study. Int J Ind Ergonom. 1993 (In Press).

26. Simmonds MJ. The reliability of palpation skills in the therapeutic professions. In: Kumar S. ed. Advances in Industrial Ergonomics and Safety IV. London Taylor and Francis 1992:665-671.

27. Threlkeld AJ. The effects of manual therapy on connective tissue. Phys Ther. 1992:72;893-902.

28. Twomey LT. A rationale for the treatment of back pain and joint pain by manual therapy. Phys Ther. 1992:72;885-892.

29. White AA, Panjabi MM. Clinical Biomechanics of the Spine. 2nd edition. Philadelphia; JB Lippincott. 1990.

CHAPTER NINE

THE RELATIONSHIP BETWEEN EXPERIMENTAL PAIN CLINICAL PAIN AND FUNCTION

Introduction

Thirty million individuals are afflicted with chronic low back problems (LBP) in the United States (1). It is estimated that 50,000,000 chiropractic and 5,259,000 physical therapist visits are made for LBP each year (32). Moreover, LBP patients comprise up to 80% of the clients in pain management centers (44) suggesting that LBP is as much a problem of pain as it is of the back (11). Despite the increase in knowledge of spinal function and dysfunction and in pain mechanisms LBP remains a costly and prevalent problem.

Accurate assessment of pain is necessary in order to make diagnoses to determine the efficacy of treatments and as a basis for making compensation awards. Assessment of endogenous pain in clinical conditions is difficult due to its subjectivity and to the complex multidimensional nature of pain. Experimental pain paradigms have been utilised in order to augment clinical pain assessment (15). However the relationship between clinical and experimentally induced pain is still enigmatic, due in part to the complexity of the pain experience. Sensory-affective relationships within the pain experience vary with different clinical and experimentally induced pains (36). For instance LBP and cancer pain have a higher affective component and a lower sensory component of pain compared to the pains of labour and to experimental pain. In the latter conditions the sensory/affective relationship is reversed (36). In addition, chronic pain patients may have altered sensitivity to acute pain and to non-painful stimuli albeit that these findings are equivocal at present.

In terms of acute pain sensitivity in chronic pain patients two distinct perceptual models have been proposed. The hypervigilant model proposed by Chapman (6) predicts that social reinforcement leads chronic pain patients to develop perceptual habits that make them hypervigilant to any noxious sensation. In contrast the adaptation model proposed by Rollman (39) suggests that the intensity of a painful stimulus is judged in comparison to other current or remembered painful experiences. Thus the chronic pain patient adapts and becomes less sensitive to pain. There is experimental support for both of these models while other investigators have reported no difference in pain perception (31).

The hypervigilant model is supported physiologically on the basis of peripheral and central neuroplasticity which leads to hyperalgesia (12,8). There is less physiological support for the adaptation model. Diffuse noxious inhibitory control (DNIC) has been suggested to account for the decreased sensitivity to acute pain in chronic pain patients. However two recent studies have failed to support this theory (35,3) suggesting that psychological factors predominate in the adaptation response.

The conflicting findings between studies may be explained by differences in clinical pathology, gender, ethnic effects and the history of other painful experiences such as childbirth (17,30,25,24,13,5,26). Differences in the methodology and the specific pain stressors used as well as the method of measuring both clinical and experimental pain will also influence the findings (50,27,3,41).

The equivocal findings in the literature suggests that both hypervigilant and adaptation models are inadequate alone. The relationship between clinical and experimental pain, -if there is one, is more complex than that explained by the simple hypervigilant/adaptation paradigm. Certainly neither model reflects the complex and dynamic nature of pain nor the individual variation of the pain experience and the complex and dynamic nature of people with pain. In many patients with chronic pain sensory affective and cognitive components can change over time. If patients are using their clinical pain as an "anchor" from which to base their judgements of experimental pain then it is reasonable to suggest that as the "anchor" changes so will the judgement. Factors such as the similarity in sensory quality of the experimentally induced pain compared to the clinical pain and the body location of experimental testing compared to the location of clinical pain may influence experimental pain measures particularly if the stressor is applied in a hyperalgesic area. In addition the duration of clinical pain and the state of exacerbation of the present pain episode may influence the results of experimental pain testing leading to changes within the individual over time. Finally the similarity in the provocative mechanism of clinical and experimentally induced pain may also influence experimental pain measures.

In general most studies using experimental pain have not assessed the impact of pain perception on function (30). Other studies which emphasise function tend not to focus on pain perception at all (37).

- **Purpose of the study**

The main purpose of this study was to measure the pain threshold in patients with LBP and compare them to a pain free control group. An additional purpose was to determine whether pain threshold in LBP patients was influenced by time or the quality of endogenous pain and the pain quality evoked by the stressor. The final purpose was to determine the relationship between clinical pain, experimental pain measures and function.

METHOD

Sample

A total of 46 subjects were included in this study 23 LBP patients in the experimental group and 23 age and gender matched subjects in the control group. The characteristics of the sample are presented in Table 9.1 there were 24 females and 22 males. The patients with LBP were recruited from an outpatient Physical Therapy Clinic. The first 23 patients referred for physical therapy with low back pain (LBP) who agreed to participate were included. Subjects with complaints of pain or abnormal sensation in their upper limbs symptomatic cervical pathology or a history of diabetes were excluded. Table 9.2 presents the pain characteristics of the experimental group.

The control group comprised a convenience sample of 23 healthy pain free subjects. They were matched by age ($\pm$ five years) and gender.

Table 9.1. Subject characteristics.

Variable	Experimental group Mean (S.D) n = 23	Control group Mean (S.D) n = 23
Age (yrs)	43.2 (12.9)	43.0 (12.4)
Height (cms)	170.1 (7.8)	168.7 (9.2)
Weight (Kgs)	76.8 (13.7)	75.1 (14.4)

Table 9.2. Clinical pain characteristics of the experimental group at initial assessment.

Variable	Experimental group n = 23
Gender	
Female	12
Male	11
Total Duration of LBP (months)	117.25 (159.69)
Duration of present LBP (months)	41.25 (112.35)
Pain intensity (0 - 10)	3.58 (2.64)
Pain affect (0 - 10)	3.44 (2.66)
Roland disability (0 - 24)	6.52 (5.15)
PT disability (0 - 10)	2.68 (2.01)
Primary pain quality	
Dull	78%
Sharp	22%
Primary pain distribution	
Back	72%
Leg	2%
Back and leg	11%

Equipment

Two mechanical pain stressors which evoked different qualities of pain were used in this study. The variable pressure dolorimeter (Pain Diagnostics and Thermography 17 Wooley Lane East Great Neck New York) is a force gauge used to apply an increasing amount of pressure through a 1.0 cm^2 surface area. The range of force that can be measured is 0 - 17 kg. The reliability and validity of this instrument has been established (42,33). The quality of pain evoked by this stressor is described as dull pressure (43).

The forceps algometer was based on a design by Burgess and Perl (4). It is a pair of forceps with an electronic strain gauge attached. The amount of force applied through a 0.1 cm^2 surface ranges between 0 and 2.7 kg. The output is measured in millivolts (see Figure 9.2). The reliability and validity of this instrument has also been established (43,28). The quality evoked by this instrument is described as sharp pinching/burning.

A 35mm tripod mounted camera, calibrated posture board with horizontal and vertical lines weights up to 5 Kg and a weight holder were used for measuring the pain threshold of controlled spinal compression. Calculation of spinal compression load was carried out using measurements made from a photograph. The measurements were entered into a computer software program for biomechanical modelling which computed the spinal compression load for pain threshold (23,7).

Clinical pain intensity and affect was measured using two numerical rating scales (NRS) one for pain intensity and one for the affect of the pain. The anchor words for each end of the intensity scale were labelled "No pain" and "Worst pain imaginable". The anchor words on the second NRS for the affect of pain NRS were: "Pain doesn't bother me" and "Pain couldn't bother me more". The reliability and validity of the NRS has been established (19,22) . The quality of clinical pain was measured by having subjects choose descriptors from the sensory domain of the McGill Pain Questionnaire (29).

Measurement of function was made using the Roland Disability Scale (38) and a timed walk. The Roland Scale is based on the Sickness Impact Profile (SIP) (2). The SIP is one of the most widely used measures of health status amongst chronic pain patients with well established reliability and validity (45,10,9,47). The shorter Roland scale is specifically focussed to measure the dysfunction associated with low back pain (38). It takes approximately five minutes to complete. The reliability and validity of this scale has been established (38,9,18). The scale is sensitive to improvement over time (38,9) and with treatment for acute low back pain (16,20).

A walk of 70 feet with two right angle turns was timed with a stop watch and used as a further objective indicator of function. Finally an NRS of disability was scored by the treating physical therapist. The anchor words were: " No disability" and "Total disability".

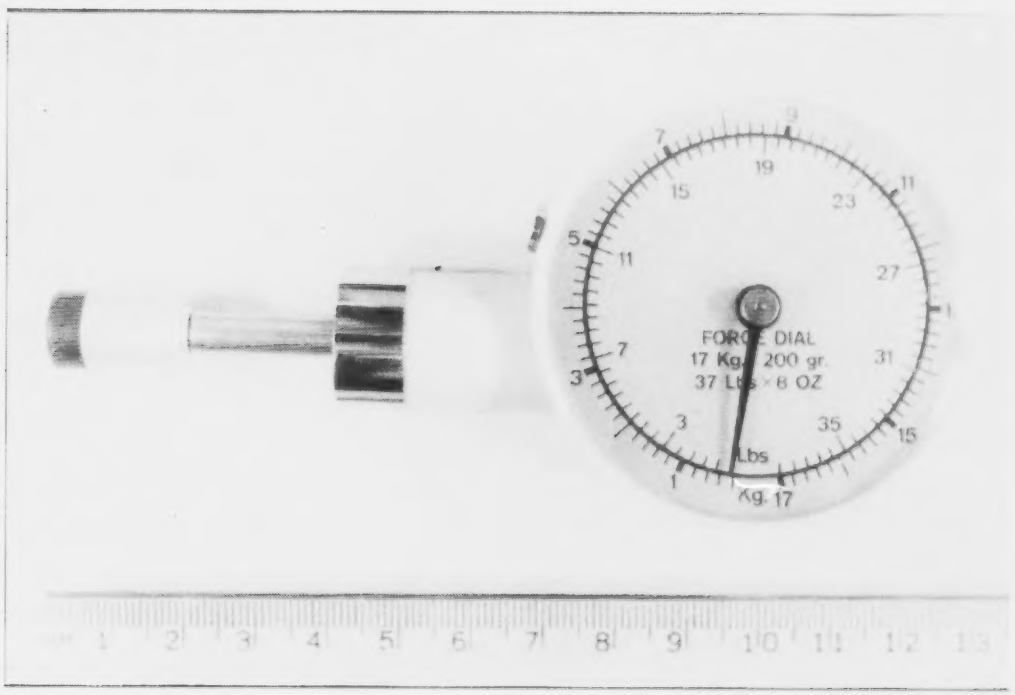


Figure 9.1. Pressure dolorimeter

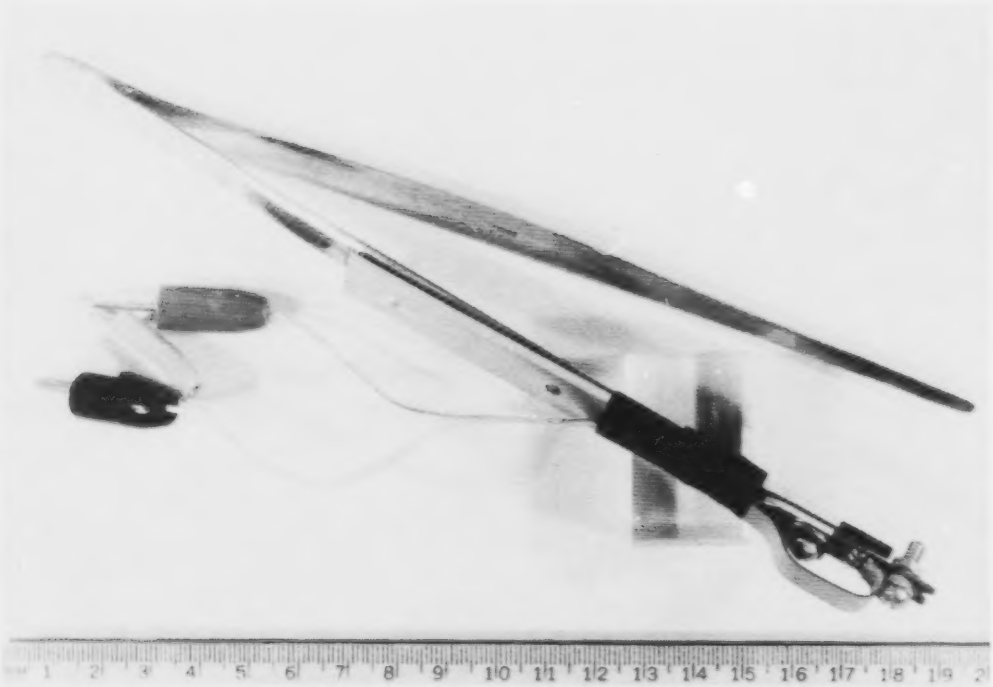


Figure 9.2. Forceps algometer

Procedure

Subsequent to the clinical interview in which the history was taken patients with low back/leg pain were informed of the purpose of the study and invited to participate. They were advised that: their decision on participation would not influence their therapy and that they could withdraw at any time without predjudice. An informed consent and a photographic release form was signed prior to participation.

The first test session was conducted within the initial week of physiotherapy (week 1). Subsequent test sessions were conducted two (week 3) and six weeks later (week 7). The treating therapist assessed the level of disability using the NRS. All other measures were carried out in a randomised manner by a trained investigator.

Anthropometric measures of weight and height were obtained in the first session only. The timed walk was conducted first. Then the experimental pain measures were obtained in random order. Experimentally induced pain threshold measured with the dolorimeter and forceps algometer was tested at the L3/L4 interspinous space and on the ulnar border of the forearm four inches distal to the olecranon process (see Figures 9.2 and 9.3). The amount of applied force with either the dolorimeter or forceps was gradually increased to the point where the stimulation was just painful. This was taken as a measure of pain threshold. There was a one minute interval between the testing of each pain threshold measure.

Spinal pain threshold measured during controlled spinal loading was obtained in the following manner Figure 9.1). Body location markers were applied to the skin at the level of the L5/S1 T12/L1 and C7/T1 spinal interspaces the tip of the shoulder the elbow and the wrist joint. The subject stood directly in front of a posture board with the feet facing forward and the side of the body towards the camera. The subject held the weight with two hands close to the body with their hands at shoulder height. The subject then reached forward with the weight until pain threshold was reached. A photographic slide was taken at the point when pain threshold occurred. An upper limit of 5 kg was set in order to avoid injury or exacerbation of symptoms.

Determination of the actual spinal load was calculated from measurements of joint angles from the photographic slide. The joint angles and anthropometric data were entered into a biomechanical software programme (23,7) in order to obtain the spinal compression load. The control subjects also performed this task but none reached pain threshold before the preset safety limit was realized. The amount of spinal compression obtained using the preset maximum weight and with the arms fully outstretched was used in the analyses.

For the timed walk the patient was requested to walk as quickly as possible along a set route. The time taken to complete the route was then recorded with a standard stopwatch. The Roland Disability Scale was administered according to the instructions on the scale. All measures were repeated

two weeks and six weeks after the first test session. The control subjects followed the same procedure except that all clinical pain and disability measures were omitted.

Data analysis

Initial analysis was by MANOVA with repeated measures on all factors. There were two 'groups' three 'pain stressors' and three 'times'. Separate one-way ANOVAs were carried out in order to examine the effects of pain stressor between groups. Within groups paired t-tests were used to determine whether there were differences between 'times'. The relationship between clinical pain experimental pain and function was tested using Pearsons correlation coefficients.

RESULTS

Group differences

A 2x3x3 (group x instrument x time) repeated measures MANOVA was run in order to test for differences between groups on spinal pain threshold measured with different instruments. There were two levels of 'group' (experimental and control) and three levels of 'instrument' (dolorimeter forceps and spinal loading) and three levels of 'time' (week 1, 3, and 7). There was a significant 3 way interaction for group x instrument x time (Pillai = .319 $F_{4\ 34}$ = 3.976 $p < .01$). The 2 way interaction for instrument x time was significant (Pillai = .351 $F_{4\ 34} = 4.605$ $p < .005$) as was the group x time interaction (Pillai = .254 $F_{2\ 36} = 6.140$ $p < .005$). The effects of group ($F_{1\ 37} = 4.11$ $p < .05$) time (Pillai = .261 $F_{2\ 36} = 6.35$ $p < .005$) and instrument (Pillai = .950 $F_{2\ 36}$ = 341.818 $p < .0001$) were all significant. The effect of location on pain threshold measured with the dolorimeter and forceps was analysed using a 2 x 2 x 2 x 3 MANOVA with repeated measures on all factors. This was two groups, two levels of location (back and arm), two instruments (forceps and dolorimeter) and three levels of time. This analysis revealed a significant effect of instrument only ($F_{1\ 37} = 291.71$ $p < .0001$).

Univariate analyses were run to determine which variables were significantly different between groups. The results indicated that walking speed was significant between groups at all times; week 1 ($F_{1\ 41} = 14.021$ $p < .001$) week 3 ($F_{1\ 41} = 10.072$ $p < .003$) and week 7 ($F_{1\ 38} = 12.711$ $p < .0001$). Spinal loading was significant at week 1 ($F_{1\ 43} = 7.88$ $p < .01$) and week 3 ($F_{1\ 43} = 3.77$ $p < .05$) but not at week 7 ($F_{1\ 43} = 1.4$ $p = .24$). Neither of the mechanical stressors for determining pain threshold were significantly different between groups. Figures 4 and 5 illustrate the means of these measures by group and location over time.

Change over time in the experimental group

Measures of clinical pain intensity pain affect and all measures of disability were analysed in order to determine change over time. A repeated measures MANOVA with 'time' as the repeated factor was utilised. Time was a significant factor for the pain measures of intensity (Pillai = .394 $F_{2\ 38} = 8.80$ $p < .001$) and affect (Pillai = .491 $F_{2\ 36} = 9.82$ $p < .0001$). It was also a significant factor for disability determined by the PT (Pillai = .699 $F_{2\ 32} = 13.85$ $p < .0001$) and the Roland scale (Pillai = .371 $F_{2\ 38} = 3.86$ $p < .05$) and for spinal loading (Pillai = .185 $F_{2\ 44} = 7.73$ $p < .001$). However, walking speed did not change over time (Pillai = .081 $F_{2\ 38} = 1.87$ $p = .169$).

For all variables the change occurred between week 1 and week 3 with no further significant change after this point. The difference between weeks 1 and 3 was: for pain intensity ($t_{19} = 3.5$ $p < .005$); for pain affect ($t_{19} = 2.9$ $p < .01$); for PT assessed disability ($t_{16} = 3.45$ $p < .005$); for Roland disability ($t_{19} = 3.20$ $p < .005$) and for spinal loading ($t_{22} = -2.71$ $p < .01$).

The effects of gender

The study sample was almost balanced in terms of gender with 24 females and 22 males. One way ANOVAs were run on the experimental group in order to determine whether there were any gender effects on clinical pain or disability measures. There were none. Spinal loading was significant but this is most likely due to body mass differences between genders.

Pain threshold measures for males and females with the dolorimeter and forceps are presented in Table 9.3. Some significant differences were revealed but only in the experimental group and only when measured with the dolorimeter.

Quality of pain

The quality of clinical pain was tested for an effect on clinical and experimental pain measures as well as on disability measures. Pain quality was categorised as primarily sharp pinching or burning in character or as a primarily dull and achy pain. Eighteen patients described their pain as primarily dull and five described it as primarily sharp.

A one-way ANOVA with the factor 'quality' with two levels was run across pain and disability variables. No differences were revealed in the dolorimeter or forceps evoked pain threshold. However pain quality did lead to significant differences between patients with sharp compared to dull pain on other pain and disability measures (see Table 9.4). Sharp pain was judged to be more intense and bothersome and also led to more disability.

Table 9.3. Means and standard deviations of experimental pain threshold measures for males and females. Dolorimeter measured in kg/cm(2 forceps measured in millivolts.

Variable	Experimental Group		Control Group	
Gender	**Females** Mean(SD) n = 12	**Males** Mean(SD) n = 11	**Females** Mean(SD) n = 12	**Males** Mean(SD) n = 11
Dol.Back				
Week 1	3.9(1.9)	5.6(2.3)*	5.0(1.9)	5.5(1.5)
Week 3	3.8(1.2)	6.7(2.3)*	5.9(3.8)	5.3(1.6)
Week 7	5.4(2.5)	6.9(3.2)	5.5(2.7)	5.5(1.8)
Dol.Arm				
Week 1	3.8(1.4)	6.7(4.2)*	5.3(2.0)	5.8(1.9)
Week 3	4.1(1.4)	5.8(3.6)	5.4(2.6)	5.4(1.5)
Week 7	4.6(1.6)	6.3(1.2)*	4.9(1.6)	6.5(2.9)
Forc. Back				
Week 1	.23(.21)	.47(.41)	.42(.12)	.50(.25)
Week 3	.28(.21)	.32(.24)	.44(.11)	.37(.12)
Week 7	.34(.20)	.37(.27)	.33(.16)	.43(.21)
Forc. Arm				
Week 1	.32(.20)	.43(.23)	.43(.12)	.50(.25)
Week 3	.35(.25)	.51(.34)	.45(.11)	.43(.18)
Week 7	.42(.28)	.55(.26)	.36(.17)	.43(.15)

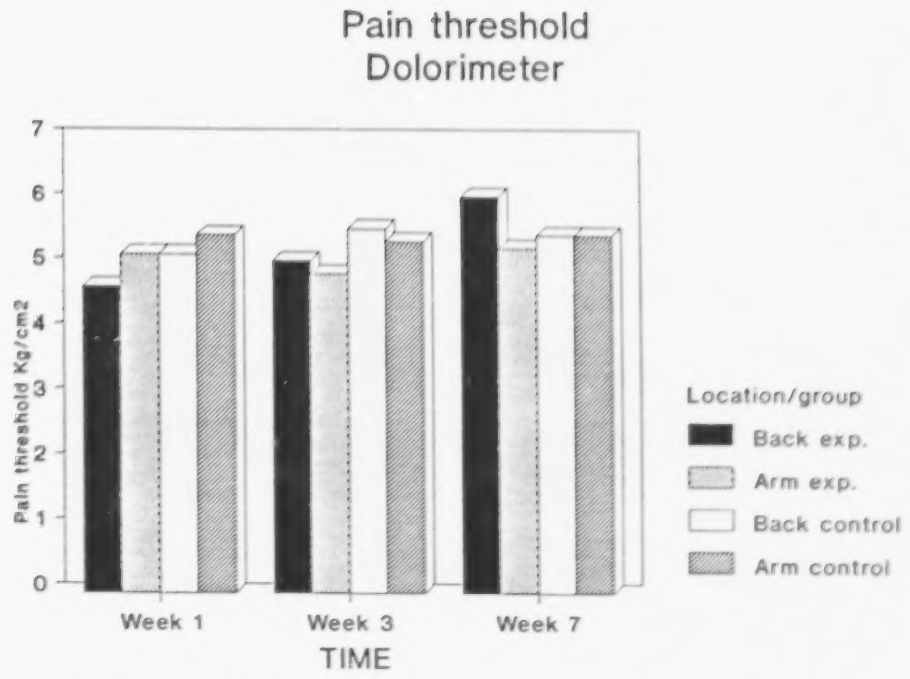
* = $p < .05$.

Disability and experimental pain.

To determine whether patients who were more disabled had lower pain thresholds patients were categorized based on their Roland disability score. Subjects in the upper 50th percentile were contrasted to those in the lower 50th percentile. A one-way ANOVA with the factor 'disability' was then run across pain and disability variables. There were no significant differences in dolorimeter of forceps evoked pain threshold measures either on the back or the arm. There was also no difference in spinal loading ($F_{1,21} = 1.90$ $p=.18$). There were differences between disability categories for walking speed ($F_{1,21} = 12.74$ $p < .001$). In addition there was a difference between subgroups for pain affect ($F_{1,21} = 5.07$ $p < .05$) whilst pain intensity almost reached significance ($F_{1,21} = 4.05$ $p = .057$).

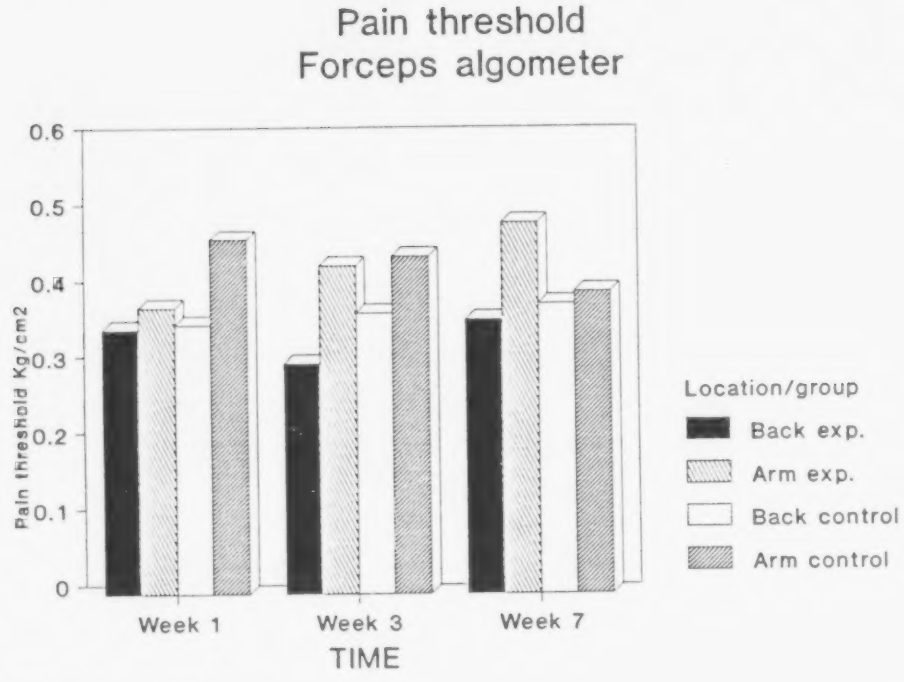
Table 9.4. Means and standard deviations for pain and disability measures in patients with sharp vs. dull pain.
F-ratios degrees of freedom and p-values for a one-way ANOVA analysis between groups.

Variable Weeks(W) 1 - 7		Sharp pain n=5	Dull pain n=18	1 way ANOVA F-ratio and (df)	Prob. of F
Pain intensity	W1	5.0(2.8)	3.7(2.4)	1.1(1 21)	.31
	W3	4.2(2.5)	2.3(2.2)	3.8(1 18)	.06
	W7	4.2(1.7)	2.1(1.8)	4.3(1 18)	.05*
Pain affect	W1	5.4(3.3)	3.4(2.0)	2.5(1 20)	.13
	W3	5.3(2.5)	2.1(2.4)	5.2(1 18)	.03*
	W7	3.5(1.3)	1.6(1.9)	3.4(1 18)	.08
Disability PT	W1	4.8(2.2)	2.5(1.4)	7.5(1 20)	.01*
	W3	5.0(2.1)	1.4(1.4)	15(1 16)	.001*
	W7	2.5(.58)	1.8(1.8)	.53(1 18)	.48
Disability Roland	W1	11(6.1)	5.3(4.2)	5.3(1 21)	.03*
	W3	10(5.1)	3.8(3.3)	11(1 18)	.005*
	W7	12(7.1)	4.9(4.9)	5.7(1 18)	.03*
Walking speed (seconds)	W1	16(4.6)	12(2.6)	7.1(1 21)	.01*
	W3	15(2.4)	12(3.0)	5.6(1 18)	.03*
	W7	16(3.4)	12(3.2)	4.7(1 18)	.04*
Spinal load (Newtons)	W1	722(226)	1132(399)	4.7(1 21)	.04*
	W3	817(322)	1217(396)	4.3(1 21)	.05*
	W7	929(335)	1280(416)	3.0(1 21)	.09



No significant differences

Figure 9.3 Mean pain threshold measured with the dolorimeter for each group on each location and on each occasion



No significant differences

Figure 9.4 Mean pain threshold measured with the forceps algometer for each group on each location and on each occasion

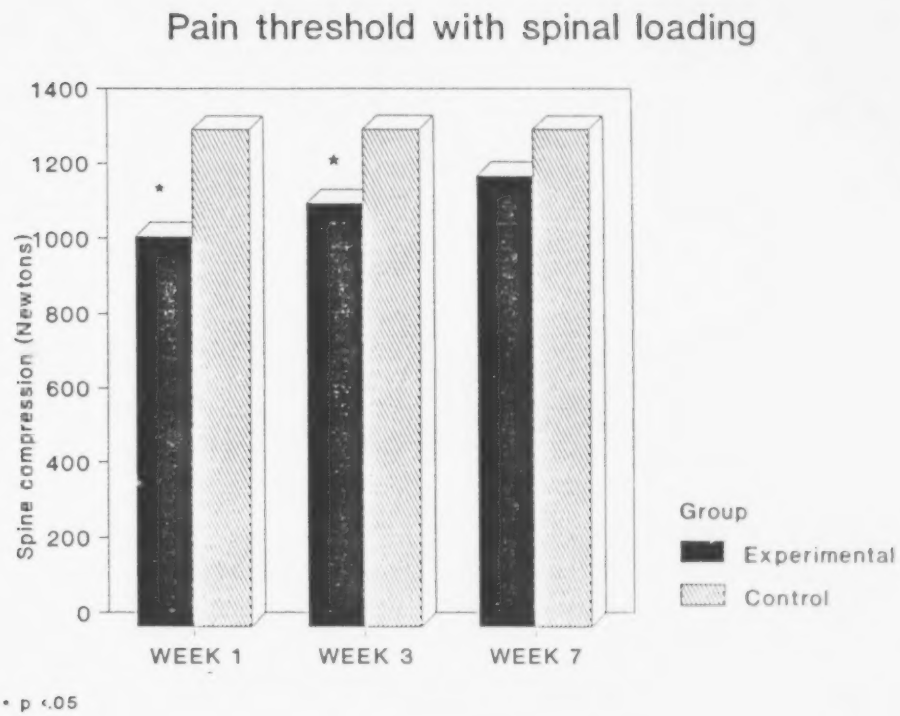


Figure 9.5 Mean pain threshold measured with controlled spinal loading for each group, on each occasion

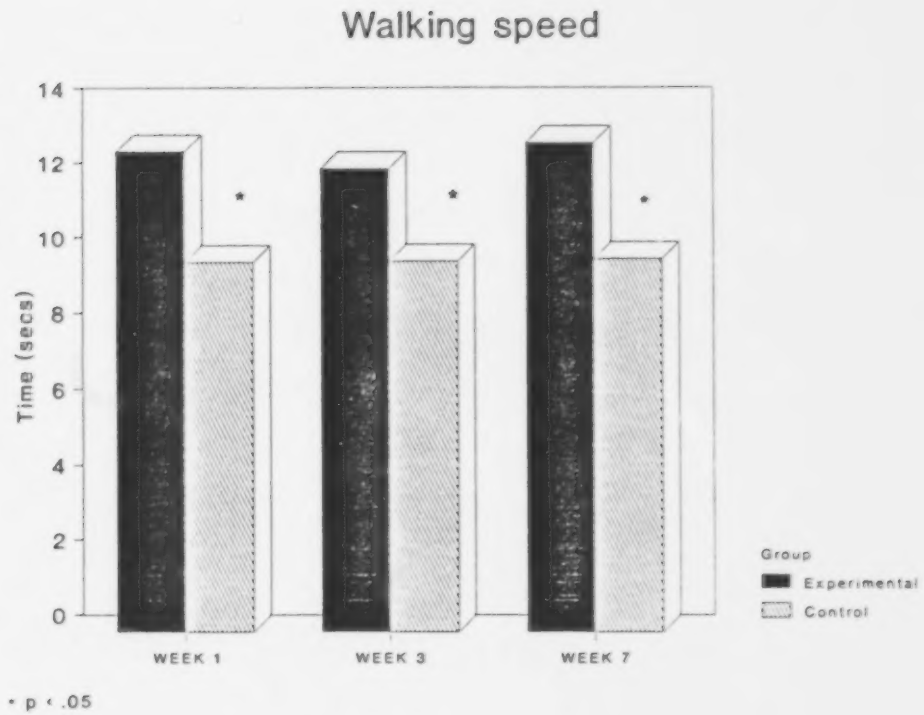


Figure 9.6 Mean walking speed for each group on each occasion

Relationship between experimental pain measures

Pearsons correlation coefficients for the dolorimeter and forceps testing conditions were run across the total subject sample as well as within each group. In the total subject sample the strongest relationships occurred between measures of pain threshold using the same instrument but tested on different locations. The highest correlation was $r = .6237$ ($p < .001$) measuring pain threshold with the dolorimeter on the back and the arm. The weakest relationship was between pain threshold values obtained with different instruments on different locations. The relationship between the dolorimeter on the arm and the forceps on the back was $r = .1322$ (n.s). A similar pattern was exhibited in the control group whilst the experimental group showed a different pattern of relationships. In this group the highest correlation was between pain threshold measured on the arm with different stressors. In general pain threshold relationships were much weaker in this group (see Table 9.5).

There were no significant relationships between experimental pain thresholds and clinical measures of pain or disability. There were however some significant relationships between clinical measures of pain and disability. These are presented in Table 9.6.

Table 9.5. Pearsons correlation coefficients between pain threshold measures in the control group (n=19) and experimental group (n=20).

Stressor/ location	Dol.arm	Dol.back	Forceps arm	Forceps back
Dol. arm	E 1.000 C 1.000			
Dol. back	E .569* C .706**	E 1.000 C 1.000		
Forceps arm	E .668** C .418	E .511 C .587*	E 1.000 C 1.000	
Forceps back	E .095 C .244	E .178 C .665*	E .120 C .574*	E 1.000 C 1.000

* = $p < .01$
** $p < .001$

Abbreviation:
dol. = dolorimeter

Table 9.6. Pearsons correlation coefficients between clinical pain and disability measures (n = 21).

Vari-able	P Int	P Aff	Dis PT	Dis Rol	Walk Spd	Spinal load
P Int	1.000					
P Aff	.7602**	1.000				
Dis PT	.4686	.3314	1.000			
Dis Rol	.4897	.5714*	.6959**	1.000		
Wlk spd	.4253	.3670	.7374**	.8836**	1.000	
Sp load	-.4328	-.1463	-.4309	-.4880	-.6018*	1.000

* = p <.01
** p <.001

Abbreviations:
P Int = pain intensity
P Aff = pain affect
Dis PT = disability judged by the PT
Dis Rol = disability measured by the Roland scale
Wlk spd = walking speed
Sp load = pain threshold measured by controlled spinal loading

DISCUSSION

Overall the results of this study suggest that the patients response to an acute noxious stimulus does not augment the assessment of clinical pain or disability. The exception to this occurs when the nociceptive mechanisms involved in endogenous pain provocation are used as a measure of pain threshold i.e controlled spinal loading. Pain thresholds measured with the dolorimeter or the forceps on either body location did not differ between LBP and control groups. In addition they were stable over time and were not influenced by the change in endogenous pain. There was a greater degree of variation in the LBP group compared to the controls and this occurred within and between test sessions but otherwise no differences between groups or locations were apparent.

The relationships between pain threshold measures did differ between groups. Within the control group most relationships were significant. The weakest relationship occurred when both instrument and location were different ($r = .24$ between dolorimeter/arm and forceps back). However the correlation coefficient for the same relationship in the LBP group is $r=.09$. The lower levels of association within the LBP group are probably due to the greater individual variability in experimental pain threshold. It is worth noting that in the LBP group the highest correlation was between the two measures of pain threshold on the arm. This implies that individual pain threshold measured on the arm is less subject to variability than on the back. The back may have been differentially sensitive to one of the stressors or alternatively the precise area of testing may have coincided with a hyperalgesic area in some patients.

Pain threshold evoked by controlled spinal loading was significantly different between groups and was not related to dolorimeter or forceps evoked pain threshold. It was more closely related to clinical measures of function and unlike pain threshold evoked with the dolorimeter or forceps spinal loading was sensitive to change in clinical pain. This suggests that controlled spinal loading was the most useful and objective indicator of clinical pain. For pressure pain thresholds there was no difference between LBP and controls. This finding is in agreement with those of Boureau et al (3). Boureau and collegues compared a heterogenous group of 53 chronic pain patients recruited from a multidisciplinary outpatient pain clinic with 17 pain free controls. They used electrical stimulation and found no difference in pain thresholds between patients and controls.

Using a similar procedure but more data manipulation Peters et al (35) reported higher pain thresholds in patients. Two groups of pain patients (chronic LBP and acute oral pain) and two control groups were tested. The authors reported that the multivariate analysis revealed no difference between the four groups for pain threshold. They also reported that there was no difference between each pain group and its control. Higher pain thresholds in both pain groups were only revealed when the data from the two control groups

was pooled and then used as a basis for comparison against each pain group separately. The results do not offer strong support for the adaptation effect reported by the authors.

In another study Peters used a one-tailed test for comparison between LBP subjects and controls. Using this test they reported higher pain thresholds in the LBP group compared to controls (34). But the experimental and control groups were not balanced for gender and the slightly higher female representation in the control group could have decreased pain threshold in the control group.

The adaptation effect has been shown by Naliboff et al (31) and Yang et al (49). They both reported higher pain thresholds and decreased discriminability in LBP subjects. In both of these studies signal detection (SD) methodology with a heat stressor was used to determine pain threshold and discriminability. However, SD methodology for pain research has been seriously challenged (40). It was interesting to find that the location of testing (back vs. arm) did not influence pain threshold even in those subjects whose primary area of pain was in the back. However the lack of group effect may be a result of individual variability. There was no evidence from this study in support of either the hypervigilant or adaptation paradigms. It is possible that sensory changes are not manifest in average minimally distressed outpatients with LBP. The patients in this study were not problem pain patients attending a tertiary pain clinic. Rather they were "average" LBP patients with symptoms severe enough to lead them to seek medical attention but with no signs of major distress or life disruption. It seems most plausible that psychological rather than physiological factors lead to changes in pain sensitivity. Moreover the psychological factors probably have to be quite marked and may be condition specific in order to reveal pain threshold changes. Pain threshold in LBP appears to be a fairly robust phenomenon. It is also true in other pain groups measured with different stressors.

For instance, Scudds (41) reported no difference in pain thresholds between patients with rheumatoid arthritis (RA) and controls. Pain threshold was measured with both variable pressure and constant pressure dolorimeters as well as with electrical stimulation. Patients with fibrositis had lower pain thresholds than controls or RA patients but this was only measurable with the variable pressure dolorimeter. Personality measures were also obtained in Scudds's study. The fibrositis group had higher levels of hypochondriasis depression and anxiety compared to the RA and control group. It is possible that the differences in pain threshold in the fibrositis group were a reflection of psychological factors rather than chronic pain.

Boureau (3) also makes this suggestion. He reported that neither pain thresholds nor electrically elicited RIII thresholds differed between chronic pain and control subjects. However he did find a difference in the rating of "unpleasantness" associated with electrically elicited pain. The chronic pain group found electrically elicited pain less unpleasant than the control group.

Boureau interpreted these findings as offering support to the adaptation model on the basis that the affective component of pain is decreased.

Clinical pain and disability

It was noted earlier that endogenous pain decreased over time. This was associated with a decrease in pain affect and an increase in measures of function. It was interesting to note that the significant improvement occurred between the first and subsequent measurement sessions, whereas no significant change took place after this time. Whether this is due to the specific or non-specific effects of physical therapy or to natural remission of symptoms can not be addressed by this study. Whitney and Von Korff (48) note that self-selection for treatment in chronic conditions occurs when there is an exacerbation of symptoms. This can result in large changes in symptomatology that are simply due to natural regression to the mean. Koes et al (21) has also shown that LBP symptoms diminish in a few weeks whether the treatment is manual therapy physiotherapy or placebo physiotherapy.

The intensity of clinical pain was not associated with other clinical measures apart from pain affect. This relationship is intuitive. The low level of association of clinical pain with disability confirms the findings of Waddell (46). He reported a correlation coefficient of r = .44 between pain and disability. The correlation coefficients between pain intensity and the Roland disability was r =.49 a similar strength in relationship was found between pain intensity and disability judged by the PT. In addition disability judged by the Roland scale and by the PT was closely associated (r=.69). Although this study did not include a stringent test of validity of this method of judging disability the results are supportive of the judgements of disability made by PTs.

Pain affect had a closer association with disability (r =.57) confirming the fairly strong relationship between psychological factors and disability. However walking speed had a stronger relationship with disability and was the best predictor of disability measured by the Roland scale (A post-hoc regression equation showed that it accounted for 77% of the variance). Walking speed was a useful measure that differed between LBP and control groups. However it was not sensitive to change.

Quality of pain

The quality of endogenous pain had no effect on the pain threshold of experimental pain. However pain quality had a much greater influence on clinical pain and disability measures. This is probably a reflection of the difference in pathology of subjects with sharp vs. dull pain. Most subjects who described their pain as primarily sharp also had complaints of leg pain indicative of nerve compromise. The intensity and affect of pain was greater in patients with sharp pain. In addition there was more compromise in physical function and on disability. This argues against the generalization of statements that

there is little correlation between pain and disability. The quality and distribution of pain influence this relationship.

Gender

In agreement with other empirical studies (5,25,24) no significant gender effect for any clinical measure of pain or disability was found. There were however gender differences in experimental pain thresholds. Similar to the findings of Lautenbacher and Rollman (27), these differences were specific to the stimulus used. In this study, a difference between genders was only present in the experimental group and only when measured with the dolorimeter.

Conclusion

In conclusion there was no support for the notion that chronic LBP leads to changes in perception of acute noxious stimuli. Experimentally induced pain thresholds were correlated with each other but not with clinical measures of pain or disability. In addition pain thresholds were not influenced by location of testing or similarity in the quality of induced pain compared to clinical pain. In addition they did not change with time or in response to decrease in clinical pain intensity. Controlled spinal loading proved to be the most useful measure of pain threshold. It was simple objective and sensitive to change in clinical symptoms.

REFERENCES

1. American Medical Associatio.n Guides to the Evaluation of Permanent Impairment, 3rd edition (revised) 1990.

2. Bergner M, Bobbitt RA, Carter WB, and Gilson BS. The Sickness Impact Profile: development and final revision of a health status measure. Med. Care 1981:19;787-805.

3. Boureau F, Luu M,. and Doubrere JF. Study of experimental pain measures and nociceptive reflex in chronic pain patients and normal subjects. Pain 1991:44;131-138.

4. Burgess PR and Perl ER. Myelinated afferent fibres responding specifically to noxious stimulation of the skin. J. Physiol. 1967:90;541-562.

5. Bush FM, Harkins SW, Harrington WG, and Price DD. Analysis of gender effects on pain perception and symptom presentation in temporomandibular pain. Pain 1993:53;73-80.

6. Chapman CR. The perception of noxious events. In: R.A. Sternbach (Ed.) The Psychology of Pain.Raven Press New York 1978:169-202.

7. Cheng C and Kumar S. A three-dimensional static torso model for the six human lumbar joints. International J. Industrial Ergonomics 1991:7;327-339.

8. Coderre TJ, Katz J, Vaccarino AL, and Melzack R. Contribution of central neuroplasticity to pathological pain: review of clinical and experimental evidence. Pain 1993:52;259-285.

9. Deyo RA. Comparitive validity of the Sickness Impact Profile and shorter scales for functional assessment in low-back pain. Spine 1986:11;951-954.

10. Deyo RA. and Diehl AK. Measuring physical and psychosocial function in patients with low-back pain. Spine 1983:8;635-642.

11. Dixon AS. Introduction. In: MIV Jayson (Ed) The Lumbar Spine and Back Pain 3rd edn. Churchill Livingston Edinburgh 1987:xi-xii.

12. Dubner R. Neuronal plasticity and pain following peripheral tissue inflammation or nerve injury. In: Bond MR, Charlton JE. and Woolf CJ.

(Eds) Proceedings of the VIth World Congress on Pain. Elsevier Science Amsterdam 1991:263-276.

13. Feine JS, Bushnell MC, Miron D and Duncun GH. Sex differences in the perception of noxious heat stimuli. Pain 1991:44;255-262.

14. Frymoyer JW. Magnitude of the problem. In: J.N. Weinstein and S.W. Wiesel (Eds.). The Lumbar Spine W.B.Saunders Philadelphia 1990:32-38.

15. Gracely RH. Methods of testing pain mechanisms in normal man. In: P.D. Wall and R. Melzack (Eds.). Textbook of Pain 2nd. edn. Churchill Livingstone London 1989:257-268.

16. Hadler NM, Curtis P, Gillings DB, and Stinnett S. A benefit of spinal manipulation as adjunctive therapy for acute low-back pain: a stratified controlled trial. Spine 1987:12;703-706.

17. Hapidou EG. and DeCatanzaro D. Responsiveness to laboratory pain in women as a function of age and childbirth experience. Pain 1992:48;177-181.

19. Jensen MP, Strom SE, Turner JA, and Romano JM. Validity of the Sickness Impact Profile Roland Scale as a measure of dysfunction in chronic pain patients. Pain 1992:50;157-162.

20. Jensen MP, Karoly P, and Braver S. The measurement of clinical pain intensity: a comparison of six methods. Pain 1986:27;117-126.

21. Klein RG, and Eek B. Low energy laser treatment and exercise for chronic low-back pain: a double blind controlled trial. Arch. Phys. Med. Rehabil. 1990:71;34-37.

22. Koes BK, Bouter LM,. vanMameren H, Essers AHM, Verstegen GMJR, Hofhuizen DM, Houben JP, Knipschild PG. The effectiveness of manual therapy physiotherapy and treatment by the general practitioner for non-specific back and neck complaints. A randomised clinical trial. Spine 1992:17;1;28-35.

23. Kremer E and Atkinson SH. Pain measurement: construct validity of the affective dimension of the McGill Pain Questionnaire with chronic benign pain patients. Pain 1981:11;93-100.

24. Kumar S and Hill D. A biomechanical model for task analysis. (Unpublished model) 1988.

25. Lander J, Fowler Kerry S, and Hill A. Comparison of pain perceptions among males and females. Canadian Journal of Nursing Research 1990:22;1;39-49.

26. Lander J, Fowler Kerry S, and Hargreaves A. Gender effects in pain perception. Perceptual and Motor Skills 1989:68;1088-1090.

27. Lawlis GF, Achterberg J, Kenner L, Kopetz K. Ethnic and sex differences in response to clinical and induced pain in chronic spinal pain patients. Spine 1984:9;7;751-754.

28. Lautenbacher S and Rollman GB. Sex differences in responsiveness to painful and non-painful stimuli are dependent upon the stimulation method. Pain 1993:53;255-264.

29. Lynn B and Perl ER. A comparison of four tests for assessing the pain sensitivity of different subjects and test areas. Pain 3;333-365.

30. Melzack R. The McGill Pain Questionnaire: major properties and scoring methods. Pain 1975:1;277-299.

31. Naliboff BD. and Cohen MJ. Psychophysical laboratory methods applied to clinical pain patients. In: C.R Chapman and J.D. Loeser (Eds.) Issues in Pain Measurement. Raven Press New York 1989:365-386.

32. Naliboff BD, Cohen MJ, Schandler SL, and Heinrich RL. Signal detection and threshold measures for chronic back pain patients chronic illness patients and cohort controls to radient heat stimuli. Journal of Abnormal Psychology 1981:90;271-274.

33. National Center for Health Statistics: Physiotherapy office visits: National Ambulatory Medical Care Survey: United States 1980-81. Advance Data from Vital and Health Statistics no.120 D.H.H.S. publication #(PHS) 86-1250. Public Health Service Hyattsville MD July 11 1986.

34. Ohrbach R. and Gale EN. Pressure pain thresholds in normal muscles: reliability measurement effects and topographic differences. Pain 1989:37;257-263.

35. Peters M and Schmidt AJM. Differences in pain perception and sensory discrimination between chronic low back pain patients and healthy controls. Journal of Psychosomatic Research 1992:36;1;47-53.

36. Peters ML, Schmidt AJM, Van den Hout MA, Koopmans R and Sluijter ME. Chronic back pain acute postoperative pain and the activation of diffuse noxious inhibitory controls (DNIC). Pain 1992:50;177-187.

37. Price DD, Harkins SW, and Baker C. Sensory-affective relationships among different types of clinical and experimental pain. Pain 19987:28;297-307.

38. Rainville J, Ahern DK, Phalen L, Childs LA, and Sutherland R. The association of pain with physical activities in chronic low back pain. Spine 1992:17;1060-1064.

39. Roland M and Morris R. A study of the natural history of back pain. Part I: Development of a reliable and sensitive measure of disability in low-back pain. Spine 1983:8;141-144.

40. Rollman GB. Signal detection theory measurement of pain. A review and critique. Pain 1977:3;187-211.

41. Rollman GB. Measurement of experimental pain in chronic pain patients: Methodological and individual factors. In: R. Melzack (Ed.) Pain Measurement and Assessment. Raven Press New York 1979:251-258.

42. Scudds RA, Rollman GB, Harth M, and McCain GA. Pain perception and personality measures as discriminators in the classification of fibrositis. Journal of Rheumatology 1987:14;3;563-569.

43. Scudds RA and Fischer AA. The use of the pressure algometerin the quantification of soft tissue pain. Phys Ther. 1988:68;777.

44. Simmonds MJ, Wessel J, and Scudds RA. Quality of pain and the effectiveness of TENS. Physiotherapy Canada 1992:44;3;35 - 40.

45. Tollison CD. Assessment and treatment at Pain Therapy Treatment Centers Program. In: C.D. Tollison Ed. Handbook of Chronic Pain Management. Williams and Wilkins Baltimore 1989:656-663.

46. Turner JA. Comparison of group progressive-relaxation training and cognitive-behavioural group therapy for chronic low back pain. J. Consult. Clin. Psychol. 1982:50;757-765.

47. Waddell G. Clinical assessment of lumbar impairment. Clinical Orthopaedics and Related Research 1987:221;110-120.

48. Watt-Watson JH and Graydon JE. Sickness Impact Profile: a measure of dysfunction with chronic pain patients. J. Pain Symp. Manag. 1989:4;152-156.

49. Whitney CW and Von Korff M. Regression to the mean in treated versus untreated chronic pain. Pain 1992:50;281-285.

50. Yang JC, Richlin D, Brand L, Wagner J, and Crawford Clark W. Thermal sensory decision theory indices and pain threshold in chronic pain patients and healthy volunteers. Psychosomatic Medicine 1985:47;5;461-468.

51. Yarnitsky D. and Ochoa JL. Studies of heat pain sensation in man: perception thresholds rate of stimulus rise and reaction time. Pain 1990:40;85-91.

CHAPTER TEN

GENERAL DISCUSSION

Overview

LBP leads to significant demands being placed on the health care system. In this thesis LBP has been reviewed from sensory and psychosocial aspects. In addition, given that PTs play a key role in the conservative management of patients with LBP, some of the fundamental skills utilised in their assessment and treatment of patients with LBP are evaluated. It is not the purpose of this general discussion to reiterate the discussion sections of each of the papers included herein. Rather a general discussion linking key findings of the studies is presented.

Physical therapy assessment of patients with LBP

An assessment is a fundamental legal and ethical requirement and must be conducted prior to any physical therapy treatment. Treatment programmes are based on and are modified by the results of assessments and reassessments. For treatment to be appropriate it is essential that assessments are reasonably accurate, are objective and are unbiased.

The influence of potentially biasing information on physical assessment findings was found to be negligible. This is a reassuring finding which suggests that as a group and in the particular research paradigm used in this study, PTs are minimally influenced by biased information.

It is possible that although compensation status was not biasing some other information may have been. The influence of expectancies could be tested using different diagnostic labels. "Diagnostic" labels such as inorganic or psychogenic back pain could be compared with discogenic back pain. However this is not a strong possibility. The fact that the control group which had no information did not differ from the other two information groups weakens the diagnostic labelling notion acting as a biasing influence. The results suggest that the information provided was either not used or else it did not influence physical assessment judgements.

Some caution must be exercised in generalizing the results of this study to the clinical situation. The fact that the PTs were knowingly participating in a research project may have influenced their clinical judgement. In addition neither palpation nor interaction with the patient formed part of the research protocol.

It is possible that interaction with the patient could have had a more profound biasing influence on assessment findings. Basmajian (1) has suggested that the main virtue of rehabilitation is the intense relationship formed between clinicians and their patients. However the quality of personal relationships can vary greatly. The "virtue" alluded to by Basmajian could just as easily be a "curse". Observation and palpation are key components of a

clinical assessment (3). So the fact that palpation did not form part of the assessment in this experiment must be addressed. Both static and motion palpation are normally utilised during physical assessments. Despite the lack of demonstrated clinical significance key bony landmarks of the pelvis are tested for position and symmetry during the initial assessment and in reassessments. However experimental results reported in this thesis suggest that the results of clinical tests based on palpation are of limited value. This is due to the magnitude of error associated with landmark location. Although spinous processes are most accurately located the amount of error is still plus or minus one spinal level. The error associated with the transverse process is even greater, arguing against specific diagnoses based on the position of transverse processes.

The accuracy of motion palpation does not fare any better. There was a significant discrepancy between what therapists thought they were doing in regards to force application and in what they did. Also in regards to the motion that therapists thought that they were feeling and in the motion that actually occurred.

The inaccuracy of force application can be explained by the lack of objective feedback presently available and the lack of quantitative descriptions of force applications. The consistent overestimation of perceived motion explains why certain "fringe" treatment techniques that purport to measure such things as cranial joint motion are embraced by some clinicians. The additional fact that there is no scientific evidence to support the techniques (6) appears to matter little.

It would appear that in some cases the expectation of motion influences the perception of motion. Walker (8) in a critical review of the sacroiliac joint has suggested that beliefs of real motion influence the perception of motion and thus dysfunction. She notes that the firmness of belief in the detection of motion may lead to overdiagnosis and overtreatment of sacroiliac joint dysfunction. Further she suggests that a strong belief in a particular preconceived theory results in disregarding information that doesn't fit with the preconception.

Preconceived notions or expectancies appear to influence very specific hard to measure tests. It is not clear whether the discrepancy is at the tactile sensory level or at the interpretive level. Error in the interpretation of information due to expectancy is plausible. The results of the study which investigated the influence of prior knowledge suggest that expectancies influence complex judgements such as prognosis or anticipated benefit from treatment, whilst there is little effect of expectancies on the relatively easy judgements of gross range of motion.

The strength of belief in one's diagnostic and treatment skills is a "double edged sword". From the negative perspective firm beliefs of a particular dysfunction lead to overdiagnosis and overtreatment. This leads to increased health care costs and can lead patients to become overly dependent on the health care system. But from the positive perspective the strength of the

clinician's beliefs in treatment efficacy increases the liklihood that treatment will be successful. If improvement of symptoms occur this will reconfirm the treatment efficacy beliefs of the clinician. Does the outcome of treatment justify the means of diagnosis and treatment? This issue warrants further discussion on a number of points.

The results of this thesis suggest that: 1. PTs perceive a need for physical therapy in the majority of people they assess no matter whether the symptoms are nonexistent, mild or severe. 2. PTs have a fairly strong belief in the efficacy of physical therapy for LBP. However, although improvement in symptoms may occur the reason for the improvement may not be due to specific treatment efficacy. The natural course of LBP and regression to the mean account for part of the improvement. In addition the placebo effects of treatment which have been reviewed in this thesis also account for part of the improvement.

Based on the results of a randomised controlled trial Koes et al (4) reported that there was no difference in effect between specific spinal mobilization therapy and general physiotherapy. Further, he suggests that the effect of both is little better than placebo. The range of individual variation in the application of supposedly similar techniques which was found in this study argues against a specific effect of spinal mobilization. The question is whether it is ethical to use treatments with effects based only on the placebo. There is no absolute or definitive answer to this question, but the costs, risks and benefits to all involved parties must be carefully evaluated.

It is clear that assessments need to be accurate and unbiased. It is also clear that PTs are fairly accurate and unbiased with gross measurements requiring little interpretation, whereas measurements that are very specific or those that require more complex judgements are less accurate and are subject to the influence of expectancies. These findings are in agreement with those of Matyas and Bach (5). Matyas and Bach reported that gross range of motion measures were judged reliably whereas specific intervertebral motion measures were not reliable. This finding is acceptable if the physical therapy treatment for the back is general. It is less satisfactory if the treatment is specific unless the PT is guided by the symptomatic responses of the patient.

Pain is one of the most frequent symptoms that lead to medical consultation. It is difficult to measure adequately because of its complexity and also its subjectivity. Although the use of experimentally induced pain has been suggested in order to augment pain assessment (7) the results of this study offered little support for this notion. Experimentally induced pain threshold had no relationship with any clinical measure. But perhaps the most significant finding was the fact that it did not differ between subjects that were pain free or in pain.

It is possible that the patients were too mild in their symptomatology to have measurable changes in sensation. The overall level of clinical pain was not that great (mean 3.58 out of a maximum of 10). It was interesting that in

this population of LBP patients receiving treatment many had relatively low levels of both pain and disability. However there were differences between groups on functional measures such as walking and in pain experienced during controlled spinal loading. This suggests that these measures are more useful in terms of measuring change. Another key point to note from this study was the differential impact of different qualities of pain. Patients who described their pain as sharp also complained of more intense pain and they had a higher level of disability. This suggests that pain quality is a useful parameter of pain to measure. The expectancies of PTs have been discussed but the expectancies of patients with LBP are also important. Patients' expectancies will influence the perception of pain the disability and the efficacy of treatment. Health related beliefs and previous experience will also determine the course of action taken by the patient during exacerbation of pain. Patients may go to the hospital emergency room; go to their general practitioner for medication or a physical therapy referral. Patients may also do nothing and wait for the exacerbation of symptoms to resolve spontaneously.

It was interesting to note that there was an improvement in pain complaints and in functional measures after three weeks of physical therapy. But it was of some concern that no further measurable improvement occurred despite ongoing treatment. Given the financial costs involved how long should treatment be continued when no measurable change is apparent? Or are we measuring the wrong things? Why do patients attend and continue to attend physical therapy? Are they compliant to the instructions of the therapist? Are therapists critically evaluating the effect of their treatment? Do patients obtain some intangible benefit from treatment that we are not measuring? Are we making them overdependent on us and at the same time diminishing their own ability to cope with a benign problem? The fact that there may be ultimate resolution in symptoms strengthens the beliefs of both therapist and patient in the efficacy of treatment, whether treatment is appropriate and necessary or not. It is clear that there needs to be a critical re-evaluation of the management of LBP. Fundamental assessment skills need to be evaluated for their reliability and validity. Methods of teaching specific skills must allow for objective feedback. Therefore we need to develop the tools that allow us to do this. Standardization of specific skills can then be obtained and they can be subjected to a critical examination of the specificity of effect. The latter is essential.

In addition assessment and treatment skills should be critically evaluated before general implementation. The enthusiasm for particular treatment trends should be tempered by critical appraisal and scientifically acceptable proof of efficacy rather than by enthusiastic testimonials. If physical therapy is to be viewed in scientifically credible terms then it has to subject its methods to scientific investigation. Further PTs should be taking an active role in the research which is critically needed. We need to determine who we can best help how we can best help and when we can best help in the management of

patients with LBP. We need to confront the question of when we are contributing to the problem rather than the solution. Waddell (7) has noted:

> our concentration on physical therapy may be contributing to the problem. We place too much emphasis on pain to the exclusion of other aspects of the illness. We overdiagnose disc lesions. We overprescribe rest. We have actually prescribed low back disability.

REFERENCES

1. Basmajian JV. Research or retrench. The rehabilitation professions challenged. Phys. Ther. 1975:55;6;607-610.

2. Graceley RH. Methods of testing pain mechanisms in normal man. In: Wall PD and Melzack R (eds). Textbook of Pain 2nd edition. Churchill Livingstone Edinburgh 1989:257-268.

3. Grieve GP. In: Common Vertebral Joint Problems Churchill Livingstone Edinburgh 1981.

4. Koes BW, Bouter LM, vanMameran H, Essers AHM, Verstegen GMJR, Hofhuizen DM, Houben JP and Knipschild PG. The effectiveness of manual therapy physiotherapy and treatment by the general practitioner for nonspecific back and neck complaints. Spine 1992:17;1;28-35.

5. Matyas TA and Bach TM. The reliability of selected techniques in clinical arthrometrics. Australian Journal of Physiotherapy 1985:31;5;175-199.

6. McKenzie RA. Mechanical diagnosis and therapy for low back pain: Toward a better understanding. In: Twomey LT, Taylor JR (Eds) Physical Therapy of the Low Back. Churchill Livingstone Edinburgh 1987:157-173.

7. Waddell G. A new clinical model for the treatment of low back pain. In: Weinstein JN and Wiesel SW (eds). The Lumbar Spine. W.B. Saunders Philadelphia 1990:38-56.

8. Walker JM. The sacroiliac joint: A critical review. Phys Ther. 1992:72;12;903-916.